CASE CONTROL STUDY ON PREVALENCE OF GESTATIONAL DIABETES MELLITUS AND THEIR OBSTETRIC OUTCOMES

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CERTIFICATE

This is to certify that the dissertation entitled "**CASE CONTROL STUDY ON PREVALENCE OF GESTATIONAL DIABETES MELLITUS AND THEIR OBSTETRIC OUTCOMES"** is a bonafide record of the work done by **Dr.V.Poomatha**, under my guidance and supervision in the Department of Obstetrics and Gynaecology during the period of her Post Graduate study at Madurai Medical College, Madurai for the degree of M.S (Branch II) Obstetrics and Gynaecology from 2011-2014.

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DECLARATION

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This is submitted to the Tamilnadu Dr.M.G.R. Medical University, Chennai in partial fulfillment of the requirements for the award of MS OG Degree Examination (Obstetrics and Gynaecology) to be held in April 2015.

Place : Madurai

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CONTENTS

INTRODUCTION

Diabetes mellitus is a group of metabolic disorder characterized by hyperglycemia resulting from defects in insulin secretion or insulin action or both. Diabetes mellitus complicates 3-5% of all pregnancies increasing the risk of both maternal and perinatal morbidity and mortality (GABBE 2003).

Incidence is going on increasing

Gestational diabetes mellitus is defined as glucose intolerance of variable severity with onset on first recognition during pregnancy. (METZGER & COUSTAIN) It complicates 3 to 5% of all pregnancies (GABBE 2003).

- Increase in the prevalence of diabetes worldwide by 40% in the past 10 years. Prevalence in 1990 4.9 percent, [Narayan et al][1] 1999 6.9 percent.
- This increased prevalence is due to obesity and life style changes such as decreased exercise. Increase in the prevalence of type II DM lead to an increasing number of pregnancies with complications.
- GDM patients are definitely associated with significantly increased maternal and perinatal morbidity. All complications associated with GDM are potentially preventable with early recognition, close monitoring and proper treatment.

- Early imprinting in inutero life of the fetus can have effects later in life. Fetal exposure of diabetes leading to childhood obesity, glucose intolerance, diabetes in adult life.

  More than Half of the women with gestational diabetes in the ensuing 20 yrs.
- There is high prevalence of diabetes mellitus and its early onset among Indians and its complications during pregnancy and its long term complications such as maternal diabetes mellitus and diabetes in offspring necessitate the importance of early screening and treatment.
- Early screening identified undiagnosed type II DM. Overt diabetes has more complications like congenital malformation, diabetic nephropathy, neuropathy, Diabetic ketoacidosis.
- Hence an appropriate method of screening has been much emphasized.
- Early diagnosis of GDM reduces maternal and perinatal mortality and morbidity.

Whereas ethnically, Indian women are more prone to develop glucose intolerance.

Indians have eleven fold increased risk. Compared to whites, necessitating Universal screening during pregnancy.

IADPSG Criteria

Hyperglycemia and Adverse pregnancy outcome(HAPO study) was designed to aid in the development of internationally agreed diagnostic criteria for GDM based on their pregnancy outcome. Glycemia was evaluated in relation to various perinatal and maternal outcomes. There was a continuous association between glucose values and perinatal outcome. This landmark multicentre International study allowed for analysis of blinder 75 gm 2 hours oral GTT data in more than 25,000 non-diabetic gravidas. International Association of Diabetes and Pregnancy Study group convened a workshop conference in 2008 and odds ratio of 1.75 was selected for the outcomes of increased neo-natal body fats, LGA and God serum C peptide greater than the 90^{th} percentile which yielded the recommended diagnostic criteria for GDM.

HAPO study was designed to determine the various levels of glucose intolerance with adverse pregnancy outcomes in women gestational diabetes. Outcomes analyzed are birth weight > 90^{th} percentile primary cesarean delivery. Neonatal hypoglycemia and cord serum c peptide levels > 90^{th} percentile IADPSG criteria based on evidence based HAPO study. Based on this study IADPSG lowered the threshold for the

diagnosis and treatment of GDM. HAPO study shows glucose concentration lower than those used in ADA correlate with perinatal complications.

- ❖ IAPSG criteria diagnose more GDM patients than routine ADA.
- ❖ IADPSG is based on pregnancy outcomes.
- ❖ We can identify more number of patient with glucose intolerance, hence we can prevent maternal & neonatal morbidity and longterm complication.

75 GRAMS OGTT –IADPSG CRITERIA

DIAGNOSTIC VALUES FOR GDM

Fasting plasma glucose	≥92 mg/dl
One hour	≥180 mg/dl
Two hours	≥153 mg/dl

Case control study was performed on 200 anti natal patients attending Government Rajaji Hospital using 75 grams glucose challenge test as screening test, 75grams IADPSG criteria OGTT as a diagnostic test and their obstetric outcomes measured.

REVIEW OF LITERATURE

Diabetes complicating pregnancy is a common endocrine disorder and was traditionally associated with poor pregnancy outcomes. The incidence of gestational diabetes is increasing the world wide. The trend towards older maternal age, the epidemic of obesity and diabetes, the decrease in physical activity and the adoption of modern lifestyles in developing countries may all be factors that contribute to this increase in the prevalence of GDM.

Since the discovery of insulin more women in their reproductive years have been able to carry their pregnancies to term ,though stillbirths, birth defects and pregnancy loss were still unacceptably high. Planned pregnancies, pre-pregnancy counseling and tight glycemic control have made it possible for these women to expect outcomes similar to women without diabetes.

Gestational diabetes (GDM) is a disorder of carbohydrate intolerance in pregnancy. Worldwide the incidence of GDM has increased and varies from 0.4% to 10% in the Western population, depending on race and ethnicity(Dornhorst et al, Coustan)[2]

In India, the incidence of GDM is much higher and ranges from 10% to as high as 17.8%. In a community-based survey Seshiah and colleagues(2008)[3] ascertained the prevalence of GDM in a South Indian

population, The prevalence of GDM in the urban, semi-urban and rural areas was 17.8%%, 13.8% and 9.9% respectively.

Over the years, there have been many controversies regarding GDM including the best screening test, diagnostic test and indeed, whether treatment does modify outcome.

Obstetrician taking care of women with gestational diabetes needs to have a good understanding of the maternal and fetal concerns arising from GDM, the management of hyperglycemia, monitoring of the fetus and timing of delivery.

Screening ,detection, counseling and follow up of diabetes in pregnancy can decrease the burden of future diabetes in women with GDM, and good glycemic control can decrease, prevent or delay the onset of diabetes in the offspring.

All complications associated with GDM are potentially preventable with early recognition of GDM, intense monitoring and treatment. Ethnically Indian women are more prone to develop glucose intolerance during pregnancy and have eleven fold increased risk compared to whites necessitating universal screening during pregnancy. Hence, an appropriate screening of GDM has been much emphasized.

A study was performed on 200 antenatal patients attending the antenatal outpatient department of Madurai Medical College Hospital to

find the incidence of GDM by 75 gms GCT followed by 75 gms OGTT using IADPSG and to correlate the abnormal results with maternal and fetal outcomes during the period from August 2013 – July 2014

DIABETES IN PREGNANCY

PRE-GESTATIONL DIABETES

Diabetes that antedates pregnancy is called Pregestational diabetes. The accepted White's classification of Diabetes in pregnancy is based on:

- The patient's condition before pregnancy
- The duration of diabetes
- Age of onset
- Complication

White's Classification of Diabetes in Pregnancy

Diabetes	Description
A	Euglycemia maintained by diet alone; diabetes may be of any duration And onset may have occurred at any age.

B Onset at age 20 years or older and duration of less than 10 years

C Onset during age 10-19 years or duration of 10-19 years

D Onset at age below 10 years, duration of over 20 years, background Retinopathy or hypertension(not PREECLAMPSIA)

F Nephropathy with proteinuria exceeding 500mg/day

R Proliferative retinopathy or vitreous hemorrhage

RF Criteria for classes R and F coexist

H Atherosclerotic heart disease clinically evident

T Prior to renal transplantation

GESTATIONAL DIABETES MELLITUS (GDM)

Gestational Diabetes Mellitus (GDM) is defined as carbohydrate intolerance (Hyperglycemia) of variable severity with onset or first recognition during the present pregnancy. The definition applies irrespective of whether or not insulin is used for treatment or the condition persists after pregnancy. It does not exclude the possibility that glucose intolerance may have antedated the present pregnancy. GDM results from sluggish first phase insulin release and in addition to excessive resistance to action of insulin on glucose utilization due to placental

hormones(Placental lactogen, progestin, prolactin and cortisol). DM women have significantly lower insuilin response at 30 and 60 minutes after oral glucose compared to normal pregnant women.. In the later half of pregnancy, insulin resistance occurs even in normal pregnancy due to placental hormones.

Insulin resistance plays a role particularly in the fasting state of the mother in ensuring that the fetus receives adequate supply of glucose by switching the material metabolism from carbohydrates to lipids. In the fed state, insulin secretion has to be augment to revert back the maternal metabolism to utilize carbohydrates. A woman with normal glucose tolerance increases her insulin secretion over and above the insulin resistance and thereby maintains her glycemic levels. Whereas a pregnant woman who is not able to increase her insulin secretion to overcome insulin resistance that occurs even during normal pregnancy, develops gestational diabetes. The aetiology and possible pathogenesis is given in Table 12. About 30% women with GDM will progress to type 3 DM in 2 to 20 years after pregnancy.

Insulin secretion in pregnancy(NGT-Normal Glucose Tolerance)

- Insulin secretion is considerable increased in both pregnant

  Women with NGT and women with GDM

- Glucose-stimulated insulin secretion is increased more in normal pregnant women than in women with GDM

- Peak plasma insulin during an OGTT occurs later in women with GDM than in women with NGT

- First phase insulin response to intravenous glucose increases more during pregnancy in women with NGT than in women with GDM

- Increases in second phase insulin responses are of similar magnitude in women with NGT and those with GDM

  Etiology and Pathogenesis of GDM: Possible Explanations

  - Autoimmune destruction of pancreatic b cells
  - Impaired b cell function
  - Increased insulin degradation
  - Decreased tissue sensitivity to insulin
    - Impaired insulin – insulin receptor binding

- Impaired intracellular insulin signaling

The risk factors for progression from GDM to type 2 DM post partum are,

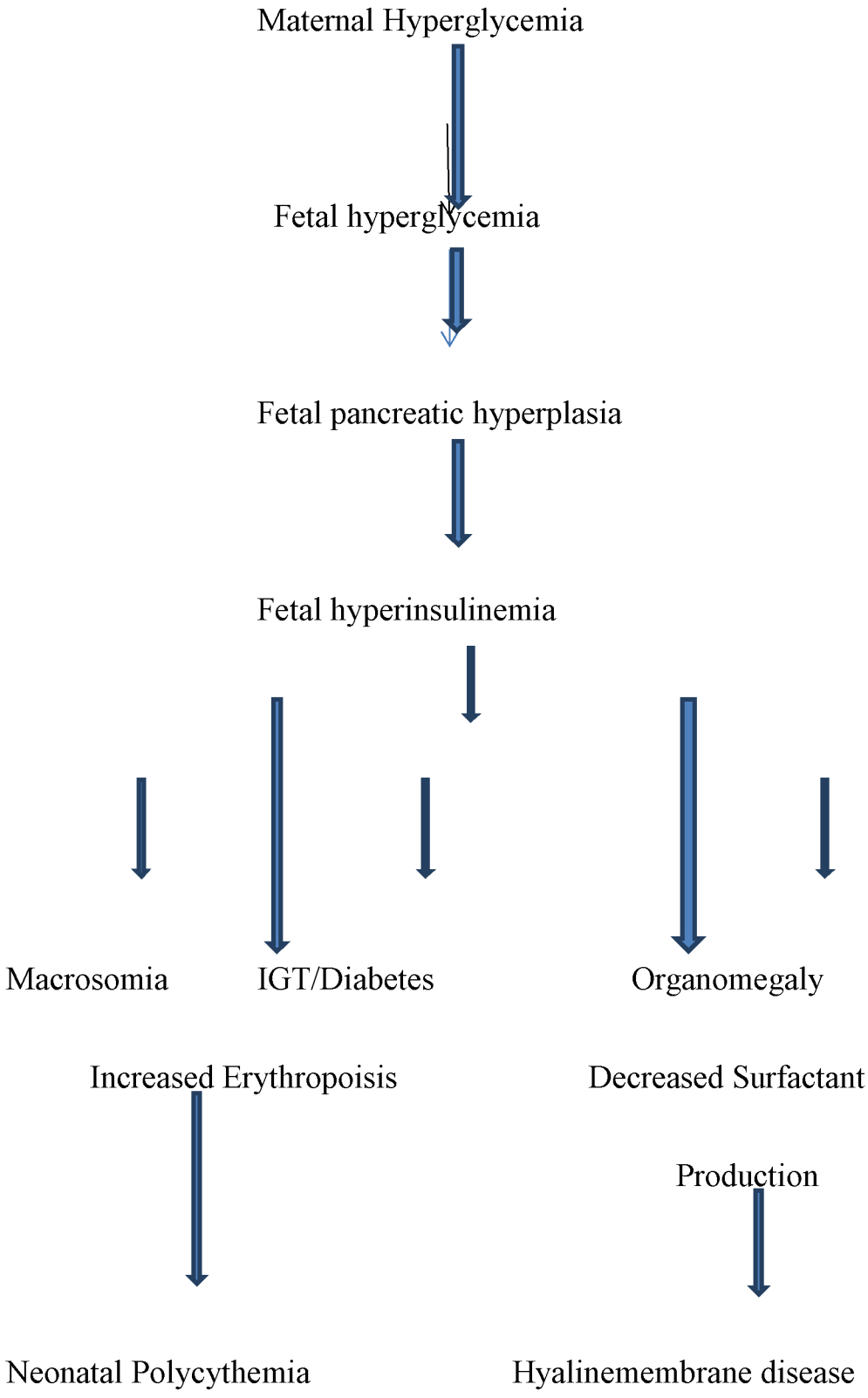
- The degree of glucose tolerance during and after pregnancy.
- Elevated fasting plasma glucose > 105 mg
- Need for insulin therapy during pregnancy
- Obesity and choice of contraception

Abnormal glucose tolerance during pregnancy is not only associated with increasing pregnancy morbidity but also increased the likelihood of subsequent diabetes in the mother. Maternal hyperglycemia has a direct effect on the development of fetal β cell mass and is associated with increased susceptibility to the development of obesity and diabetes in the offspring

Effect of maternal fuels on fetal development; The hyperglycemia-hyperinsulinism hypothesis of Pedersen has been modified to include contributionsof other maternal fuels besides glucose that are also responsive to maternal insulin. All of these can influence the growth of the fetus, maturation of fetal β cells and increased insulin secretion. Within this formulation, growth will be disparately greater in insulin-sensitive than in insulin-insensitive tissues on the fetus.

The effect on the offspring is independent of other generic factors. As such GDM has implications beyond the index pregnancy, identifying two generation(mother and her offspring) at risk of future diabetes. The recognition of glucose intolerance during pregnancy is more relevant in the Indian context as Indian women have 11 fold increased risk of developing GDM compared to White women. It is important to detect these GDM cases because if unrecognized, pregnance may end in perinatal death and fetal wastage.

PEDERSON'S HYPOTHESIS



GESTATIONAL GLUCOSE TOLERANCE(GGI)

The term 'impaired Gestational Glucose Tolerance (IGGT)' is used by few authors to indicate pregnant women whose 2 hr PG>120 mg/dl but < 140 mg/dl. It may be appropriate to use the term 'Gestational glucose intolerance (GGI)' instead of Impaired gestational glucose tolerance. Further, quiet frequently we come across, labeling any abnormal value n the OGTT not meeting the diagnostic criteria of GDM as IGT. The use of this term 'IGT' during pregnancy may be confusing, as this terminology is also being used in non-pregnant adult with 2 hr PG >140 mg/dl. This level is also applied to diagnosed GDM by WHO criteria. Hence, it may be prudent to label 2 hr value>140mg/dl as GDM and a 2 hr PG value > 120 mg/dl as GGI. The term IGT should not be used to denote any abnormal value during pregnancy. The figures suggested as follows are easy to remember.

With 75 gm OGTT (WHO criteria):

	In Pregnancy	Outside Pregnancy
2 hr≥ 200mg/dl	Diabetes	Diabetes
2 hr≥ 140 but≤199 mg/dl	GDM	IGT
2hr≥120 but ,130 mg/dl	GGI	Normal

Women with GGI and without overt gestational diabetes was associated with a significantly increased incidence of cesarean section, pre eclampsia and macrosomia. An elegant study using innovative computer based technology indicate patients with one elevated blood glucose value during formal glucose tolerance testing have higher blood glucose values under ambulatory conditions. Furthermore, these elevated ambulatory glucose values were significantly correlated with fetal macrosomia

MATERNAL AND FETAL ADAPTATIONS IN PREGNANCY

A new structure arises denovo during pregnancy. It develops and matures till it expelled at the completion of the gestational period. The metabolic adaptations that occur during pregnancy are to accommodate a rapidly growing tissue transplant, the conceptus. For its own normal growth and development, the conceptus brings about alterations in maternal fuel metabolism and hormones. The placenta facilitates embryogenesis, growth, maturation and survival of the fetus. It has the capacity to synthesize steroid and peptide hormones and to modulate and transport maternal fuel to the fetus.

FUEL METABOLISM IN NORMAL PREGNANCY

The fuel metabolism during normal (non-diabetic) pregnancy is characterized by

- Facilitated insulin action during the first half of pregnancy and

- Diabetogenic stress during the second half of pregnancy.

Early Weeks of Gestation

In the early weeks of gestation certain hormonal changes occur,

- Increase in fasting insulin concentration
- Increase in glucose stimulated insulin release (which reaches a peak at the

  $18^{th} – 20^{th}$ week)
- Increase in serum levels of estrogen and progesterone which induces beta cell hyperplasia.

These hormonal changes result in an increased elaboration of insulin (hyperinsulinemia) and heightened sensitivity to insulin. Insulin, being an anabolic and anti-catabolic hormone favors the following

- Tissue glycogen storage,
- Prevents production of glucose from the liver
- Increase peripheral glucose utilization.

The net effect of these anabolic changes is a decrease in fasting blood glucose by 10% compared to non-pregnancy fasting level. The other reason for the decrease in the fasting plasma glucose is due to increase in plasma volume in early gestation and increase in feto placental glucose utilization .

Carbohydrate Metabolism in Early Pregnancy (upto 20 weeks)

Hormonal Alteration	Effect	Metabolic change
Estrogen and Progesterone Beta-cell hyperplasia and insulin secretion	↑ Tissue glycogen Storage ↓ Hepatic glucose Production ↑ Peripheral glucose utilization ↓ Fasting Plasma Glucose	Anabolic Due to sex Steroids + Hyperinsulinaemia

Later Weeks of Gestation

During the later half of pregnancy, the facilitated insulin action continues. At the same time, there is an increased elaboration of placental chorionic somato – mammotorphin (Human Placental Lactogen, HPL), prolactin and cortisol. These surges in counter hormones result in insulin resistance. Stress on the carbohydrate metabolism (diabetogenic stress) and due to this, maternal insulin sensitivity is reduced approximately by 50%. Extra insulin compensates for the 50% reduction in the responsiveness of peripheral tissues to

insulin action due to placental hormones (Fig-1). In a normal pregnant woman, first and second phase insulin response increases approximately three fold by the third trimester and is associated with maternal B cell hypertrophy and hyperplasia.

Overall, the metabolic alterations under the influence of insulin and placental hormones facilitate anabolism during feeding and catabolism during fasting. As the pregnancy advances, the plasma glucose during fasting continues to be low, due to constant removal of glucose by the fetus since the fetus is a continually feeding boarder in an intermittently eating host, the mother. The lower level of FPG is also attributed to a fall in circulating amino acids particularly alanine which is needed for gluconeogenesis, a situation of "substrate deficiency syndrome". The fetus removes both glucose and amino acids from the maternal circulation, the former by facilitated diffusion and the later by active transport. Hence during pregnancy, maternal nitrogen is conserved (for the fetal use) by sparing protein and relying as little as possible on proteins and carbohydrates as substrate. The maternal metabolism shifts rapidly to catabolism when exogenous fuel in not available, using fat as the fuel source. Placental hormones help in this metabolic shift by producing ketogenesis and a state of "accelerated starvation". The metabolic changes which should normally occur after 72 hours of food

deprivation in a non-pregnant state occurs within 18 hours during pregnancy.

Glucose and a variety of substrates act in a coordinated manner to regulate insulin and glucagon secretion. Normal alpha cell function serves to protect against hypoglycemia (aminogenic stimulation – gluceneogenesis) and to minimize prandial glucose excursions. During fed state, the levels of insulin and glucose are higher and more prolonged. Following a glucose load, glucagon is more readily suppressed during pregnancy that in the non-pregnant state. The combination of enhanced beta cell response (extra insulin) to glucose and preserved alpha cell response to amino acids leads to 'facilitated anabolism' from 'mixed meals' : The "extra" insulin that is secreted in response to meal blunts the gluconeogenic potential of glucagon during the immediate postprandial hyperglycemia and so "spare" ingested amino acids for fetal access. Contrariwise, after disposal of the carbohydrate, the responsiveness of the alpha cell to the persistent hyperaminoacidemia could stimulate enough gluconeogenesis to prevent reactive hypoglycemia in the mother

Carbohydrate metabolism in late pregnancy (20 to 40 weeks)

Hormonal change	Effect	Metabolic change
↑ HPL ↑ Prolactin	"Diabetogenic" ↓ Glucose tolerance Insulin resistance ↓ Hepatic glycogen Stores ↑ Hepatic glucose Production	Facilitated Anabolism during feeding Accelerated starvation during fasting ↓ Ensure glucose and AA to fetus

In short, facilitated anabolism in the fed state and accelerated starvation in the fasted state characterize the maternal fuel adaptations during pregnancy. These hormonal and metabolic changes are geared towards facilitated anabolism under the actions of insulin throughout pregnancy. However, in the second half of the pregnancy, insulin resistance and diabetogenic stress due to placental hormones necessitates compensatory increase in insulin secretion. Hence the clinical expression of gestational diabetes occurs, where this compensation is inadequate, usually during the second half of pregnancy.

: Changes in Maternal Metabolism During Normal Pregnancy
Decreased fasting plasma glucose level Increased post prandial plasma glucose level Increased fasting and post prandial plasma insulin levels β – cell hypertrophy and hyperplasia Decreased insulin sensitivity (insulin resistance due to placental hormones) Enhanced lipolysis

PATHOGENESIS OF GLUCOSE INTOLERANCE DEVELOPING DURING PREGNANCY

I) GENETIC FACTORS

The relative importance of genetic factors has not yet been established. Increasing maternal age and obesity are significant contributing and compounding factors.

II) GESTATIONAL FACTORS

a) Islet secretion

In gestational diabetes, during OGTT, the early insulin release is sluggish (Fig-2). They have significantly lower insulin response at 30 and 60 minutes after oral glucose, compared with glucose tolerant controls. Due to this, as the pregnancy progresses the time to reach the maximum glucose

concentration increases. The mean time to reach the peak plasma glucose level is 55 minutes at 38 weeks of gestation compared to 34 minutes in the non-pregnant state. There is no diminution in the biologic activity of circulating insulin and the alpha cell function in gestational diabetes.

b) Insulin resistance and hormones of gestation

- In about 20% of gestational diabetics, the sluggish early insulin secretion cannot be demonstrated. It may be that in these individuals, there is an increased elaboration and/or heightened sensitivity to one or more of the gestational counter hormones (like human placental lactogen (HPL) leading to decreased insulin sensitivity (Fig-3).
- The post receptor defects in the insulin signaling cascade appears to be a cause for the decreased insulin sensitivity in both pregnant women with normal glucose tolerance and gestational diabetes compared to weight matched non pregnant controls.
- Insulin receptor substrate (IRS-1) expression is decreased in all pregnant women compared to non pregnant controls. The down regulation of IRS-1 protein parallels the decreased ability of insulin in inducing further steps insulin signaling cascade. The factors affecting IRS-1 function in the signaling cascade is due to cytokine tumor necrosis factor (TNF-α)

- In other works the pathophysiology of gestational diabetes has been related to excessive insulin antagonism by the pregnancy contra insulin factors. When maternal insulinogenic compensation is inadequate to offset these factors, gestational diabetes will supervene.
- Further when gestational diabetics how had reverted to normal glucose tolerance following delivery and women with normal glucose tolerance during pregnancy were given prednisolone or HPL in the postpartum period and challenged with Oral Glucose load (OGTT), the former failed to increase plasma insulin levels above those in the latter, despite greater hyperglycemia. Thus the decreased β cell function that occurs in GDM women may be the indication for the future susceptibility to diabetes.

FUEL METABOLISM IN DIABETIC PREGNANCY

The effect of diabetic pregnancy of fuel metabolism is due to under utilization of exogenous fuels in the fed state (facilitated anabolism reduced) and over production from endogenous sources in the fasted state ('hyper' accelerated starvation). The first sign of pregnancy in a pre GDM (particularly in Type-1 DM) as early as the first week of gestation and even before nausea or vomiting sets in, may be early morning fasting ketonuria. This should not be considered as ketoacidosis.

CONSEQUENCES OF DIABETES ON THE FETUS

Consequences of the changes in fuel metabolism during pregnancy on the fetal development revolve around 'maternal hyperglycemia and fetal hyperinsulinemia'

Pregnancy is considered as a tissue culture experiment implicating that placenta and fetus develop in an incubation medium that is totally derived from maternal fuels consisting of glucose, amino acids and lipids. The glucose traverses the placenta by facilitated diffusion and amino acids by active transport and enter the fetal circulation (Fig-5).

The recent concept is that the placental glucose transport is dependent on the glucose transporter (GLUT) family and GLUT 1 is the principal transporter which is located in the syncytiotrophoblast. GLUT 1 is present both on the microvillus and basal membranes. With the advancing gestation there is a two to three fold increase in the expression of syncytiotrophoblast transportation. The role of GLUT 3 and GLUT 4 remains speculative. Amino acids are actively transported across the concentration gradient form the mother to both fetus and placenta via energy requiring amino acid transporters. These processes are regulated by insulin and hence any disturbance in the secretion and action of insulin will influence the whole nutrient composition to which fetus is exposed and may lead to feta hyperinsulinemia. The abnormal mixture of maternal

nutrients gain access to the developing fetus and modify phenotype gene expression in newly forming cells and thereby causing permanent short and long term effects in the off springs. Accordingly the fetal and neonatal complications occur when the fetus is exposed to the abnormal fuel mixture during different periods of gestation.

Fetal Problems Associated With Maternal Hyperglycemia By Trimester

First Trimester	Second Trimester	Third Trimester
Malformations Growth Retardation Fetal Wastage Placental insufficiency	Hypertrophic Cardiomyopathy Polyhydramnios Erythraemia Respiratory distress syndrome Preclampsia Fetal loss Low IQ	Hypoglycemia Hypocalcaemia Hyperbilirubinemia Macrosomia Hypomagnesemia Intraurerine death

FIRST TRIMESTER

In the first trimester, the exposure to abnormal mixed nutrients during organogenesis (first 6-8 weeks of gestation) may cause spontaneous miscarriage, intrauterine growth retardation and malformations

Studies have shown that the maternal metabolic abnormalities are the most important cause for the increased risk of malformations in diabetic pregnancies.

The mechanisms suggested for the teratogenic effect in the early post implantation stage of the embryo are,

- Disruption of normal functioning of the yolk sac which regulates nutrient transport from maternal plasma to embryo during early neural tube development.
- Diffusion of intracellular myoinositol with resultant disruption of arachidonic acid and prostaglandin metabolism
- Oxidative metabolism and generation of free oxygen redicals that may be toxic to the embryos and
- Glucose induced mutations in embryonic DNA.

Type and Timings of Malformation in infants of Diabetic Mothers

Type of Anomaly	Timing of Lesion (Weeks Post conception)
Skeletal	
Caudal regression	3
Spina bifida	6
Neural	
Anencephaly	4
Myelocele	4
Hydrocephalus	5
Cardiovascular	
Dextrocardia	4
Conus arteriosus defects	5
Ventricular septal defects	6
Renal	
Renal agenesis / hypoplasia	6

If this fuel mediated teratogenesis is to be avoided, then excellent control of maternal metabolism must commence before conception and must be maintained during the first eight weeks, a critical period when

many women may not be aware that they are pregnant. Preventive medicine necessarily starts before conception reflecting the importance of pre-pregnancy counseling. Supplementation with folic acid dose (0.4 mg), myoinositol and antioxidants may play a role in the prevention of malformations and lower the ratio from 7.5 to 0.8%.

SECOND TRIMESTER

- The formation and the development of brain cells takes place in the second trimester. Hyperglycemia during this trimester alters the behavioral , intellectual and psychological pattern in childhood.
- Insulin is detectable in the fetal pancreas as early as 9^{th} week after conception. β cell growth and replication are regulated by nutritional insulin secretogogues such as glucose, mannose and essential amino acids.
- Human studies have shown an increase in pancreatic fetal cell mass and insulin secretion in fetuses of poorly controlled diabetic women of 16^{th} week of gestation ad both these abnormalities increase throughout second trimester until 26^{th} week of gestation.
- This priming of β cell in mid gestation may account for the persistence of fetal hyperinsulinemia throughout the pregnancy and the risk of acclelrated fetal growth even when mother achieves a good metabolic control in later pregnancy.

THIRD TRIMESTER

- Maternal hyperglycemia in the third trimester causes proliferation of fetal adipocytes, muscle cells and pancreatic β cells and neuro endocrine systems and they form the base for macrosomia and for the development of obesity, IGT and type 2 diabetes in later life.
- Still birth in diabetic pregnancies is still unexplained, although both maternal hyperglycemia and fetal macrosomia are associated.
- Mechanisms implicated are fetal hypoxia, acidosis, hypokalemia leading to dysrhythmias and placental dysfunction and competition for essential nutrients.
- In an unexplained stillbirth, the possibility of undiagnosed GDM must be considered and at autopsy the fetus may have islet cell hyperplasia (in the absence of rhesus problem) and increased interstitial tissue in the testis or lutenization of the theca interna of the ovary.

NEONATAL COMPLICATIONS :

Early pregnancy complications

Congenital Malformations

Three major complications of neo-natal period for infant and diabetic mother is congenital defect, hypoglycemia, asphyxia. Gabbe[4] reported decrease in still-birth rate and change in rate of congenital

malformations have moved major malformations to first place among all causes of perinatal mortality for IDM. Poor metabolic control during organogenesis as evidenced by elevated first trimester glycohemoglobin values. Elevated glycosylated haemoglobin values is associated with increase in risk of malformations.

Malformation among IDM Ranges between 6-9%.

50% of Perinatal mortality in IDM due to congenital malformation.

It is related to the degree of glycemic control during organogenesis. HBA1C reflects glycemic status of the patient.

Spontaneous abortion

Spontaneous abortion rate in controlled diabetic patients was between 10-17%.

It is related to glycemic control in Early trimester [Joslin Diabetes Center].Patients who had first trimester HbA1C values more than 6 standard deviations had a two-fold increased risk of malformations.

Congenital Malformation

Caudal regression syndrome is more specific. 200 fold more common in diabetic offsprings.

Hypoglycemia :

Hypoglycemia in the first few hours of life is seen Iin 30 to 50% of infants of diabetic mother. 50% of hypoglycemia occurs among

macrosomic infants. With near physiological control of maternal glucose, the incidences 5 to 15% .

Due to the endogenous hyperinsulinemia and suppression of endogenous glucose production, the Infant of the diabetic mother (IDM) is at increased risk of hypoglycemia at 1 to 3 hours after birth. About 50% of the hypoglycemia babies may remain asymptomatic. Twitching of the limbs, hypotonia, tachypnoea and rarely seizures in severe hypoglycemia are the clinical presentations. Hypoglycemia is defined as blood sugar level less than 45 mg/dl in any infant regardless of gestational age. Once hypoglycemia is confirmed, IV glucose as an initial bolus of 200 mg/dl must be given and is followed by a continuous glucose infusion at 8 mg/kg/mt. The factor mainly protective against fetal hypoglycemia is the optimal control of maternal hyperglycemia especially during the third trimester and during labor. It has been shown that a mean maternal plasma glucose > 105 mg/dl during the last four hours of labor in a diabetic mother leads to a higher incidence of neonatal hypoglycemia.

Hypocalcemia :

About 25% of the IDMs may present with serum Calcium of <7 mg/dl and this may remain mostly asymptomatic and is usually detectable during the 2^{nd} or 3^{rd} day of the birth. Asphyxia and prematurity, operating through elevated Cortisol, induces Vitamin D antagonism at the intestinal

level. Respiratory distress and fetal metabolic acidosis may result in calcium being shifted from intracellular to extra cellular pools and reversal of this shift during correction of the acidotic event may procedure hypocalcaemia. Hypomagnesemia may coexit and may require correction.

Respiratory Distress Syndrome (RDS) :

Incidence of respiratory distress syndrome among IDM is 5 to 6 fold higher than the incidence among infants of non-diabetic women. Glucocorticoids,thyroxine,promotes Type 2 pneumocyte proliferation and hence surfactant production . Insulin and androgen inhibit surfactant production.

RDS is observed in about 5% of Infants of Diabetic Mothers (IDM). In vitro studies indicate that insulin antagonizes the stimulatory effects of crotisol of fibroblasts to induce the synthesis of Fibroblast-Pneumonocyte Factor (FPF), which in turn inhibits type II cells and phosphatidyl Choline production.

Measurement of Phosphatidyl glycerol alone or in combination with the lecithin phosphatidyl choline may be a more reliable indicator of lung maturity in diabetic pregnancies than the Lecithin; Sphingomyelin ratio alone.

- Prophylactic steroids to accelerate the lung maturity may be indicated if the L : S ratio is < than 2:1.

- Such obstetric situations, requiring steroids or β sympathomimetics drugs (E.g.Salbutamol) may worsen the diabetic control and calls for frequent monitoring of blood sugar and correction with soluble insulin.

Polycythemia ;

It is relatively common in Infants of Diabetic Mothers (IDM). This is mostly due to hypoxic stimulus of the placental insufficiency and elevated Glycohemoglobin. Over transfusion from the large placenta of diabetic pregnancy may also contribute. The resultant hyper viscosity may induce congestive heart failure and vascular thrombosis accounting for the increased risk of renal vein thrombosis in these infants.

Hyperbilirubinemia :

Neo-natal jaundice has been reported in 25 to 53 % of pregnancies complicated by pre-gestational diabetes and 38% of pregnancies in women with GDM. Jaundice is increased in macrosomic babies. Severe hyperbilirubinemia may be observed independent of polycythemia. HbA1C values in late pregnancy are significantly elevated in mothers of hyperbilirubinemic infants.

This common abnormality is due to increased bilirubin production and decreased life span of the RBCs with glycosylated cell membranes. Hepatic conjugation of bilirubin may be impaired due to immature liver.

Macrosomia :

ACOG defines macrosomia as birth weight greater than 4.5 kg.Delivery of infant weighing greater than 4.5 kg occurs ten times more often in women with diabetes compared in the population of women with normal glucose tolerance. Fetal macrosomia complicates 50% of pregnancies with GDM and 40% of pregnancies complicated by Overtdiabetes.

Neonates weighing more then 4 kg are considered to be marcosomic. The Indian consensus is that new born baby's weight >3.5 kg should be considered as macrosomia. Macrosomic babies have considerable greater shoulder/head and chest/head differences and are prone to shoulder dystocia. Fetal hyperinsulinemia per se is accompanied by excessive transfer of nutrients to the fetus and external somatic growth. This phenomenon usually manifest around 28^{th} week gestation. The fetal insulin has a central role in fetal growth and development sround the last 10 weeks of gestation. Meticulous control of maternal metabolism (substrate concentration) tends to normalize the fetal growth to a certain extent.

Two typer of macrosomic infants can be identified :

1) **Consitutional macrosomia**, which represents the genetic drive to growth – his infant is LGA (> 90^{th} percentile) already in the second trimester and will continue to grow on its own growth curve during pregnancy.

2) **Metabolic macrosomia**, which represents diabetic fetopathy as a result of abnormal glucose metabolism – this infant is characterized by increased liver and spleen size (greater abdominal circumference) and normal head size (head circumference). In addition, this infant suffers from an enlarged heart and increased subcutaneous fat.

Perinatal Mortality

Major improvements in the care of pregnant diabetic women have reduced the perinatal mortality rate but it still remains higher than that for the offspring of non-diabetic women. A review of available studies confirms the over-all risk of perinatal mortalities related to the degree of metabolic control.

Other metabolic abnormalities:

Hypocalcemia ,Hypomagnesemia are common among IDM and usually asymptomatic. Routine screening is not recommended . These conditions should be considered if a baby remains unusually jittery despite normal glucose.

Unexplained fetal demise

Unexplained fetal demise is relatively unique to pregnancies complicated by overt diabetes. Infants die before labour usually at 35 wks or later. They are large for gestational age.

Possible mechanisms are

1. HbAIC concentration is higher in diabetic women .Glycosylated hemoglobin tightly bound to oxygen, release oxygen less well – decreased oxygenation to placenta.
2. Fetus hyperinsulinemia causes increased oxygen requirement of fetus. Oxygen supply is unable to keep pace with demand which causes fetal hypoxia and acidosis.
3. Maternal hyperglycemia causes osmotically induced villous edema and impaired fetal oxygen transport.
4. Placental insufficiency, when associated with advanced diabetes and vascular complications.

Preterm Birth

Single most important cause of pre-term birth in diabetic complicating pregnancy is Preeclampsia. There was a strong relationship between diabetic nephropathy and pre-term delivery. Canadian study by YANG et al[5] – Incidence of preterm birth is 28%. Five-fold increase

compared with that of their normal population Overt diabetes is a risk factor.

9 percent of women with diabetes[Sibai et al 2000][6] delivered before 35 wks. Compared with 4.5 percent of nondiabetic women.

7 percent underwent indicated preterm delivery.

Joslin diabetes Centre - Incidence of preterm birth was 26%

CONSEQUENCES OF DIABETES ON THE PREGNANT MOTHER

Complications of diabetes in pregnancy occur almost exclusively in pre gestational diabetic women.

1) Hypoglycemia

Hypoglycemia may occur in the first trimester of the pregnancy. This is due to combination of physiological adaptation, attempt for strict control and the nausea of early pregnancy.

2) Retinopathy

Prevalence of diabetic retinopathy is strongly related to the duration of disease. Pregnancy is associated with progression of diabetic retinopathy .Degree of severity of progression is related to both pre-existing retinopathy and degree of metabolic control.

Background diabetic retinopathy (BDR) can develop or worsen during pregnancy. It is not a risk for vision and usually regresses post partum. If

BDR is already present, may progress to proliferative diabetic retinopathy (PDR). Therefore, It is essential to perform periodic ophthalmic examination and in a few photocoagulations may be necessary. The pregnant women with poor glycemic control and with hypertension are at an increased risk of developing PDR. These risks can be minimized by instituting pre conception control of diabetes and hypertension.

During pregnancy with regression postpartum. Some among them are likely to

Diabetic Keto Acidosis

It occur in 1% of Diabetic pregnant patient [Garner 1995].[7] It is most common in type I diabetes mellitus fetal loss is 20% [Pederson's & Cocuorhers][8]. Pregnant women develop ketoacidosis even with lower blood glucose level than nonpregnant.

Nephropathy

Prevalence of nephropathy among diabetic pregnant women is as high as 14%.

5% overt diabetes pregnant women will have renal complication. The risk of worsening diabetic nephropathy depends on baseline renal function and the degree of hypertension. Diabetic women with microalbuminuria may develop albuminuria during pregnancy with regression in postpartum period. Some among them likely to develop

preeclampsia symptoms. If the intitial renal function is impaired in pregnancy. Almost 50% of them are likely to show further decline in renal function.Women with nephropathy develop complications like Iugr ,preterm birth, preeclampsia.Women with nephropathy develop complications like pre-term labour, IUGR

Preeclampsia

Hypertension may be induced or aggrevated by pregnancy. It is a major complication that most often forces pre-term delivery in diabetic women. Special risk factors for preeclampsia includes any vascular complications and pre-existing proteinuria.

Factors aggrevating preeclampsia in a diabetic women are vascular complications, preexisting proteinuria, with or without chronic hypertension.

Most of the preterm delivery in diabetic women is due to preeclampsia.

Perinatal mortality is 20 fold increased in preeclamptic women with diabetics preeclampsia is related to blood glucose value [Temple et al][9].Temple and co-workers (2006) studied HbA1C levels at 24 weeks with Type 1 diabetes. They found that preeclampsia was related to glucose control.

Diabetic Gastropathy

It is a form of diabetic neuropathy. symptoms nausea, vomiting, nutritional problems, difficulty with glucose control.

This condition severely exacerbates nausea and vomiting. The drug such as cisapride or mosapride may give relief.

Infections

In diabetic pregnancies all type of infections are increased.Stalmer reported 80% of women with type 1 diabetes develop atleast one infection during pregnancy.Pregestational diabetes is associated with two to three fold increased risk of post operative wound infections(Stalmer)[10].**Common infections are** Candida vulvovaginitis, urinary infections, respiratory infection pelvic infections.

Polyhydramnios

Polyhydramnios is associated with poor control of glucose.

Reason for polyhydramnios is not certain. Probable reasons are

1. When the fetus passes more urine than usual due to hyperglycemia.
2. Amniotic fluid glucose irritates the amnion to produce increased amounts of liguor (Dashe et al).[11]

Malpresentations and premature labour are more common in polyhydramnios [Sibai et al. 2000)[12]

SCREENING FOR GESTATIONAL DIABETES MELLITUS

Universal screening detects more cases and improves maternal & fetal outcomes. Incidence of diabetes is increasing among Indians. There is increased incidence of both pregestational and gestational diabetes. Studies have shown that maternal metabolic abnormalities are the most important cause of increased risk of maternal & fetal complications. To avoid maternal and fetus complications excellent control of maternal metabolism must commence before conception and must be maintained throughout the pregnancy. Oral glucose tolerance test carried out during each trimester is the ideal procedure. This unfortunately is not possible in centers with high birth rates. The usual recommendation for screening is between 24 to 28 weeks. The recent concept is to screen for glucose tolerance in the first trimester itself.

Screening

All aspects of diagnosis and management of gestational diabetes including whom to screen, how to screen, when to screen is controversial.

ACOG & ADA recommends selective screening.

ADA – 2009 recommendation

Low risk

Age $\leq$ 25 yrs

Normal body weight

No family history

No history of abnormal glucose metabolism

No history of poor obstetric outcome

Not a member of ethnical group with high prevalence of diabetes.

[Native American, Asian, Pacific islanders]

Indian are ethnically more prone to develop diabetes. Indians have eleven fold increased risk necessitating universal screening.

Screening for GDM

Urine Glucose

The renal threshold for glucose is lowered during pregnancy.

Cause

1. There is eightfold increase in glomerular filtration of glucose.
2. Intermittent tubular defect in glucose absorption during pregnancy.

When the tubular maximum for glucose is exceeding glycosuria occurs. In pregnant women glucose is found in urine at lower blood glucose levels than in nonpregnant. This lowered renal threshold during pregnancy makes glycosuria less specific for detection of GDM. It is not used as diagnostic test. If glycosuria is 2+ or more GTT should be advised. [Gribble RK]

Blood Glucose

Blood glucose levels normally lowers during pregnancy.

It makes the non pregnant blood glucose criteria unsuitable for pregnancy.

There is no optimal approach to screening for gestational diabetes, despite more than 40 yrs of research.

Prevalence of type 2 diabetes in general is increasing in younger population.

It lead to an increasing number of pregnancies with diabetes. [Ferrara et al 2004][13].

Many women with gestational diabetes are likely to have undiagnosed type II. [feig 2002[14]].

Incidence of diabetes complicating pregnancy has increased 40 percent between 1989 and 2004. [Getahum et al 2008][15].

Age adjusted prevalence tripled from 14.5/1000 women in 1991 to 47.9/1000 in 2003 [Baraban et al 2003][16].

In the Mini environment of uterus, the early imprinting have effects later in life [Saudek 2002].[17]

Fetus is exposed to maternal hyperglycemia which leads to fetal hyperinsulinemia. Fetal hyperinsulinemia causes increase in fat cells and

leads to obesity and insulin resistance in childhood [Feig Palda 2002]. There events lead to impaired glucose tolerance and diabetes in adult.

As the prevalence of diabetes is in increasing trend. Oral glucose tolerance test carried out during each trimester is the ideal procedure it is not possible in centers with high birth rate.

All the pregnant women should undergo OGTT.

Particularly the high risk population and ethnically vulnerable populations like Indians.

Universal / Selective Screening

American Diabetes association recommends selective screening. Selective screening is applicable for women belonging to low ethnic group with low prevalence of GDM.

Indications for selective screening ADA 2009.

1. Age > 25 yrs
2. Obesity (when prepregnancy BMI > 25)
3. Family history of diabetes
4. Previous history of unexplained perinatal loss
5. Intrauterine death
6. Large for gestation age infant
7. History of congenitally malformed infant.

8. Members of ethnical Group of high prevalence of diabetes. [Native American, Hispanic American, Asian, Pacific islander].

Indians are coming under high ethnic group.

ADA recommends selective screening

Screening if performed between 24 to 28 hours in women who are not known to have glucose intolerance earlier in pregnancy.

Two Step Procedure

First a 50 gm of oral glucose challenge test is done.

It is followed by diagnostic 100 gm of oral glucose tolerance test.

GCT

Glucose levels are measured 1 hr after 50 of GCT without regard to time of day or previous meal.

If the cutoff is set as ≥ 140mg / dl 80 percent of women with GDM are identified.

Cut offs is set as ≥ 130 mg / dl 90% of women with GDM are identified.

100 gm of OGTT by carpenter and coustan method

Time	100 gm OGTT
Fasting	≥95 mg/dl [5.3 mmol / L]
1 hour	≥180 mg/dl [10 mmol / L]
2 hour	≥155 mg/dl [8.6 mmol / L]
3 hour	≥140 mg/dl [7.8 mmol / L]

When two or more values are met or exceed this value GDM is diagnosed.

WHO criteria.

OGTT is performed after over night fasting by giving 75 gm of glucose. Plasma glucose is measured at fasting and 2 hrs.

WHO criteria (75gms OGTT)

	FASTING	2 h (mg / dl)
Gestational diabetes	≥110	≥140

Screening by DIPSI method with 75 gm of glucose

Advantages

1. Seldom a pregnant woman visiting the antenatal clinic for the first time comes in the fasting state if she is asked to come on another day in the fasting state she may not return. So it is preferable to perform the diagnostic test at the first prenatal visit itself in the Indian population.

   This procedure assumes clinical relevance as WHO criteria based on 75 gm glucose.

   This procedure serves as both screening and diagnostic procedure.

IADPSG Criteria

Hyperglycemia and Adverse pregnancy outcome(HAPO study) was designed to aid in the development of internationally agreed diagnostic criteria for GDM based on their pregnancy outcome. Glycemia was evaluated in relation to various perinatal and maternal outcomes. There was a continuous association between glucose values and perinatal outcome. This landmark multicentre International study allowed for analysis of blinder 75 gm 2 hours oral GTT data in more than 25,000 non-diabetic gravidas. International Association of Diabetes and Pregnancy Study group convened a workshop conference in 2008 and odds ratio of 1.75 was selected for the outcomes of increased neo-natal body fats, LGA and God serum C peptide greater than the 90^{th} percentile which yielded the recommended diagnostic criteria for GDM.

HAPO study was designed to determine the various levels of glucose intolerance with adverse pregnancy outcomes in women gestational diabetes. Outcomes analyzed are birth weight > 90^{th} percentile primary cesarean delivery. Neonatal hypoglycemia and cord serum c peptide levels > 90^{th} percentile IADPSG criteria based on evidence based HAPO study. Based on this study IADPSG lowered the threshold for the diagnosis and treatment of GDM. HAPO study shows glucose

concentration lower than those used in ADA correlate with perinatal complications.

- ❖ IAPSG criteria diagnose more GDM patients than routine ADA.
- ❖ IADPSG is based on pregnancy outcomes.
- ❖ We can identify more number of patient with glucose intolerance, hence we can prevent maternal & neonatal morbidity and longterm complication.

75 GRAMS OGTT –IADPSG CRITERIA

DIAGNOSTIC VALUES FOR GDM

Fasting plasma glucose	≥92 mg/dl
One hour	≥180 mg/dl
Two hours	≥153 mg/dl

Overt diabetes:

A diagnosis of overt diabetes can be made in women who meet any of the following criteria.

- Fasting plasma glucose ≥ 126 mg/dl
- HbA1c ≥ 6.5%
- Random plasma glucose ≥ 200 mg/dl

HBA1C:

HBA1C is glycosylated hemoglobin.its concentration is proportional to the blood glucose level. It is useful in retrospectively assessing the metabolic control in previous 8-12 weeks. There is potential relationship between first trimester metabolic control and risk for major malformation.

Hb A1c Standard deviations about mean	Percentage
<6.1	3.7
6.1-9.0	5.2
9.1-12.0	8.2
12.1-15.0	32.2
>15.0	41.7

MANAGEMENT

The important predictor of fetal outcome either in pregestational or Gestational diabetes is the glycemic control attained immediately before and during pregnancy. The plasma glucose level of normal pregnant women is less than 90 and 120 mg/dl respectively during fasting and non-fasting states. Hence, the best fetal outcome can be expected by maintaining the mean plasma glucose level around 105 mg in a pregnant diabetic woman.

MANAGEMENT OF DIABETES IN PREGNANCY

Pre gestational type 1 and type 2 diabetic women

The congenital malformation remains the leading cause of mortality and serious morbidity in infants of mother with type 1 or type 2 diabetes, inspite of advancement in understanding pregnancy metabolism and treatment. Studies have established association between elevated material glucose during embryogenesis and high rates of spontaneous abortions and major malformations in newborn. Clinical trials also have established preconception care to achieve tight glycemic control during first trimester have resulted in striking reductions in malformations. Unfortunately unplanned pregnancy occurs in a considerable number of women with diabetes resulting in fetal mortality and morbidity.

The perinatal morbidity attributable to conditions such as macrosomia and metabolic disorders remain relatively high in women who develop glucose intolerance of any degree with onset or first recognized during pregnancy(Gestational Diabetes Mellitus,GDM). Yet another observation was that in pregnant with one elevatged blood glucose during formal glucose tolerance test have abnormal glucose values under continuous ambulatory glucose monitoring. These elevated ambulatory glucose values were significantly correlated with fetal macrosomia. Thus the foetus of pre-gestational diabetic women, gestational diabetic women or women with any degree of abnormal glucose tolerance during pregnancy is at risk of developing either congenital malformation or morbidity in the form of macrosomia.

To minimize the occurrence of lethal malformations, pre-gestational counseling is essential. The pregnant women with diabetes need standard care throughout pregnancy. The goal for glycemic management in the pre conception period and during the first trimester should be to obtain the lowerst A1c test level possible without undue risk of hypoglycemia in the, would be mother.

Practical self-management skills are essential for attaining good glycemic control in preparation for pregnancy and during pregnancy.

1. Use of appropriate meal plan
2. Self monitoring of blood glucose
3. Self administration of insulin and adjustment of insulin doses
4. Treatment of hypoglycemia (patient and family members)
5. Incorporate safe physical activity
6. Development of techniques to reduce stress and cope with the denial.

All these measures are applicable in women with gestational diabetes also.

Preconceptional care.

60% of pregnancies among pregestational diabetes are unplanned. So, most of the diabetic women frequently begin their pregnancy with suboptimal glucose control (Kim and Colleagues 2005)[18]

Glycosylated Haemoglobin measurement is useful to assess early metabolic control. Optimal preconceptional glycosylated haemoglobin values as those within three standard deviations of the normal mean. The most significant risk for malformation is with levels exceeds 10 percent (ACOG 1994)

Early pregnancy loss and congenital malformations are common in infants of overt diabetic mothers.

Optimal medical care and education are recommender for optimum control of glucose in periconceptional glucose levels levels (ADA)

Preprandial 70 to 100 mg/dl

Postprandial glucose levels < 140 mg/dl (1hr)

< 120 mg/dl (2 hr)

Folate 400 mg/day given in periconceptional period decrease the risk of neural tube defect

Medical Nutrition Therapy

Landon and associates (2009)[19] describe the benefits of dietry counceling and monitoring in women with Gestational diabetes.

ADA recommends nutritional councelling diet provides an average of 30 Kcal/kg/d based on prepregnancy body weight. This is for non obese women.

Obese women with BMI > 30kg/m2 benefit from a 30 percent caloric restriction. Obese patient are monitored with weekly tests for ketonuria because ketonuria is associated with impaired psychomotor development in off spring. For underweight women – 40 Kcal/dg/day. Insulin was required to achieve glucose control in obese women.

Diet composition 55% of diet should be composed f carbohydrates, preferably of low glycemic index. The protein should be 20% of calories. Total fat intake should be less than 25% saturated fats should be less than 10% diet taken as three meals and three snacks daily.

Exercise

Exercise during pregnancy reduced the risk of gestational diabetes. [Dempsy and co-workers][20]

Brankston and associates (2004)[21]

Exercise diminished the need for insulin therapy in obese with diabetes. Light exercises and especially exercise of the upper part of the body is safe in pregnancy. ACOG (2001), Bang 1993 – results.[22]

Exercise improved cardio respiratory fitness in pregnancy.

Oral hypoglycaemic agents

ACOG (2001) has not recommended oral hypoglycemic agents. Lange et al [23](2000)- normoglycemic level were achieved equally well with both insulin and oral hypoglycemic agents.

Glyburide

Kremer (2005), Jacobson 2005– glyburide was thought not to cross the placenta

Moretti et al 2008[24] – No increased perinatal risk with glyburide.

Hypoglycemia was less frequent than insulin.

however Hebert et al (2008)[25] – found umbilical cord concentrations were half that of the maternal concentrations in women treated with glyburide.

ACOG recommends further randomized trails regarding the use of glyburide.

Metformin

Metformin was found to reduce the incidence of gestational diabetes in women [Glueck 2004] [26]who are taking metformin throughout the pregnancy.

Metformin was known to reach the fetus (Harborne 2003)[27] – it should be discontinued once pregnancy is diagnosed.

Metformin treatment for GDM limited to clinical trials with long term infant followup [Fifth international work shop conference]

Rowan et al [2008][28] – Metformin was not associated with increased perinatal complications.

46% of women in metformin required insulin supplements.

4% of women treated with glyburide required supplemental insulin.

Insulin therapy

Insulin is indicated when medical nutritional therapy fails.

ACOG (2001) recommendation.

If fasting $\geq$ 95, 2hr postprandial $\geq$ 120 -Insulin started

It is preferable to start with premix insulin 30/70 (short acting / intermediate acting)

Total insulin dose per day can be divided into 2/3rd in the morning, 1/3rd in the evening.

If postprandial glucose is still not under control – consider rapid acting insulin analogue. It is safe during pregnancy.

Multiple injections of regular short acting insulins are preferable. Most popular regimen four times daily regimen. It includes three injections of regular short acting insulin before three major meals and a bed time injection of Intermediate acting Insulin. Preneal insulin dose can be increased depending on the corresponding post meal values of blood sugar. NPH increased if fasting glucose is high. Patients who is on insulin should be aware of Hypoglycemic symptoms.

Insulin pump therapy can be used as an alternative .One approach to insulin administration is the use of the continuous, subcutaneous insulin infusion pump(the insulin pump') in order to more closely approximate physiologic insulin release. Randomised trial of pump versus intensive conventional insulin therapy in pregnant women with diabetes failed to reveal any difference in metabolic control or perinatal outcomes(Coustan et al)[29]

Glucose Monitoring

Self monitoring using a glucometer and capillary blood glucose has to be encouraged. Women using daily self monitoring had fewer macrosomic infants compared to women evaluated during hospital visits only [Hawkins and colleagues (2009)][30]

Postprandial surveillance was superior to preprandial surveillance.

In postprandial surveillance – blood glucose control was significantly improved and was associated with fewer cases of neonatal hypoglycemia, less macrosomia, fewer casarean delivery.

Goal values in mg/dl for adequacies of glycaemic control in pregnancy [ACOG 2005]

HbA1C	≤	6
Fasting	≤	95
1 hr postprandial	≤	140
2 hr postprandial	≤	120
2-6	≥	60
Mean		100

Obstetrical Management

Antepartum

1. Nuchal translucency is assessed at the 11-14 weeks.
2. Detailed anomaly scan is performed at 18-20 uhs.

3. Fetal echo has to be done.
4. Growth scan have to be performance in the 28, 32 and 36 weeks.
5. Women who require insulin therapy should undergo antepartum fetal surveillance from 32uhs.

TIMING OF DELIVERY

Gestational diabetes, who are well controlled with diet, who do not require insulin, seldom require early delivery.

They can be followed till 40 weeks.

In GDM patients on insulin pregnancy is usually terminated 38 weeks. Antenatal corticosteroids should be administered to mother in case of preterm delivery.

Type of delivery

Diabetes itself is not an indication for caesarean section.

Vaginal delivery may be allowed if there are no maternal or fetal complications. When the fetus is not excessively large, and the cervix is considered favourable.

ACOG (2001) suggested cesarean delivery in women with a sonographically estimated fetal weight $\geq$ 4500. Cesarean delivery rates were 50 to 80 percent (Gabbe 1997) [31]

Elective induction reduced the rate of shoulder dystocia from 2.2 to 0.7 percent [Conway and langer 1996][32]. Continuous CTG monitoring in labour is mandatory.

Postpartum evaluation

Postpartum follow up is recommended because 50 percent of women with gestational diabetes develops overt diabetes within 20 years. Insulin therapy during pregnancy, particularly before 24 weeks, is a powerful predictor of persistent diabetes [dacus and coworkers 1994]. If patient had fasting hyperglycemia during pregnancy, than diabetes is more likely to persist postpartum. Recurrence rate of GDM subsequent pregnancies was 40 percent [Holmes et al 2003[33]].

Women with Gestational diabetes are at increased risk for cardiovascular complications associated with metabolic syndrome [Dyslipidemia, Hypertension, abdominal obesity]. [Pallardo and colleagues 1999][34]

ADA 2007 recommendations for postpartum followup – using 75gm 2hr OGTT 6-12 uhs postpartum – for postpartum classification of glucose metabolism.

1 yr postpartum, then annually, trainnually, and again before the next pregnancy.

Insulin management during labour. (ACOG 2005)

Usual dose of insulin is given as bedtime.

withhold the morning dose of insulin.

Intravenous infusion of normal saline to be started.

Once the active labour begins, or glucose levels decreases to 70mg/dl, 5percent dextrose infusion started at a rate of 100-150 ml/hr.

Glucose levels are checked hourly.

If the glucose level exceeds 100 mg/dl. Regular (short acting insulin) is started as IV infusion at a rate of 1.25 u/hr.

Insulin therapy

Insulin is indicated when medical nutritional therapy fails.

ACOG (2001) recommendation.

If fasting $\geq$ 95, 2hr postprandial $\geq$ 120 -Insulin started

It is preferable to start with premix insulin 30/70 (short acting / intermediate acting)

Total insulin dose per day can be divided into $2/3^{rd}$ in the morning, $1/3^{rd}$ in the evening.

If postprandial glucose is still not under control – consider rapid acting insulin analogue. It is safe during pregnancy.

Multiple injections of regular short acting insulins are preferable. Most popular regimen four times daily regimen. It includes three

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AIMS & OBJECTIVES

1. To assess the Prevalence of Gestational Diabetes Mellitus
2. To estimate the maternal and Perinatal outcome of Gestational Diabetes Mellitus and to compare with normoglycemic pregnant women.

MATERIALS AND METHODS

Design of Study : Case – Control

Duration of Study: 1 year

Place of Study : Department of Obstetrics & Gynaecology, Govt. Rajaji Hospital, Madurai Medical College, Madurai.

INCLUSION CRITERIA : Women aged 18 – 40

EXCLUSION CRITERIA : Known case of type 1 & type 2 diabetes mellitus

Auto immune disorders – includes systemic lupus

Erythematosis, thyroid disorders

Hyperprolactinemia

Patient on ovulation induction drugs

METHODS

200 Pregnant women with above criteria are selected for this study.

All the 200 pregnant women will undergo Glucose Challenge Test with 75gms of glucose and venous blood sampling will be done after 2 hours of glucose intake irrespective of their previous diet.

Glucose Challenge Test ≥ 140 mg is considered positive.

Those who are positive will under go confirmative oral glucose tolerance test with 75gms of glucose using IADPSG CRITERIA. This test defined as positive if any one of the hourly plasma glucose levels meet or exceed the following values.

Blood Sample	**IADPSG Criteria**
Fasting	≥92 mg/dL
1-hour	≥180 mg/dL
2-hour	≥153 mg/dL

HbA1c will be done to patient positive for GDM.

Other parameters noted – Hb, Blood Glucose, Urea, creatinine, Urine albumin sugar deposits, ultra sonogram, doppler study of fetus

Pregnant women diagnosed to have Gestational Diabetes Mellitus will be treated and followed. Outcomes measured and the outcomes will be compared with normoglycemic patients. Following outcomes are measured.

Primary Outcomes

Fetus -	Prematurity	Hypoglycemia
	IUD	Hypocalcemia
	Macrosomia (wt > 4kg)	Hyperbilirubinomia
	IUGR	
	oligohydramnios	
	Congenital anomalies	
	Shoulder dystocia	
Mother -	PIH	
	Antepartum Hemorrhage	
	Oligohydramnios / polyhydramnios	
	Preterm labour	
	Caesarian deliveries	
	Instrumental deliveries	

RESULTS AND ANALYSIS

Table 1

Incidence

According to our study the incidence of gestational diabetes mellitus in 200 patients selected during one year period at Madurai Medical College.

Total Number of Cases Screened	200
Number of cases detected having GDM	8
Incidence of GDM	4%

Incidence of GDM in our study 4%

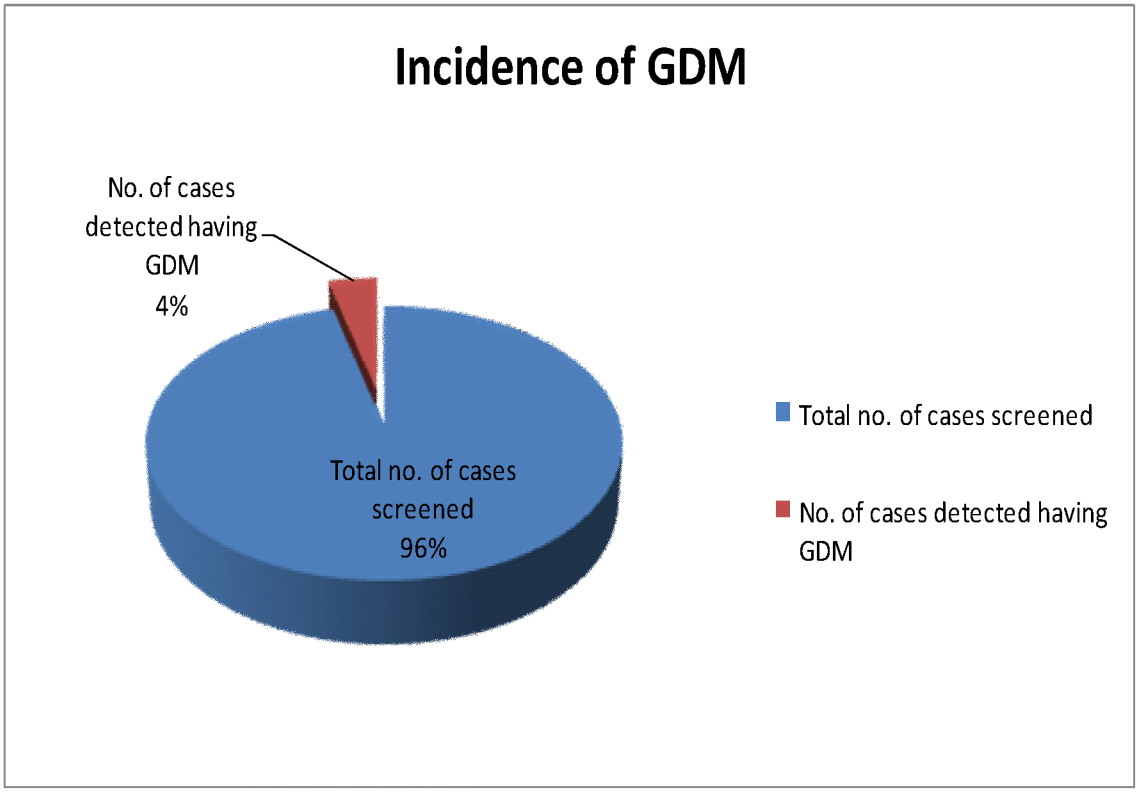

Incidence of GDM
No. of cases detected having GDM 4%
Total no. of cases screened 96%
Total no. of cases screened
No. of cases detected having GDM

Table 2

Age Distribution

Age in Years		Screening Positive for GDM	Screening Negative for GDM	Total
18-20 yrs	No. of Pts	0	32	32
	Percentage		16.6%	
21-25 yrs	No. of Pts	1	101	102
	Percentage	12.5%	52.6%	
26-30 yrs	No. of Pts	5	39	44
	Percentage	72.5%	20.31%	
31-35YRS	No. of Pts	2	15	17
	Percentage	25%	7.8%	
36-40yrs	No of pts	Nil	5	5
	Percentage		2.6%	

Chi Square value : 5.98 P Value : 0.014 Significant

Age of patients ranged from 18-40 years. Majority of patients with GDM were in the age of 26-30 years 72.5%.

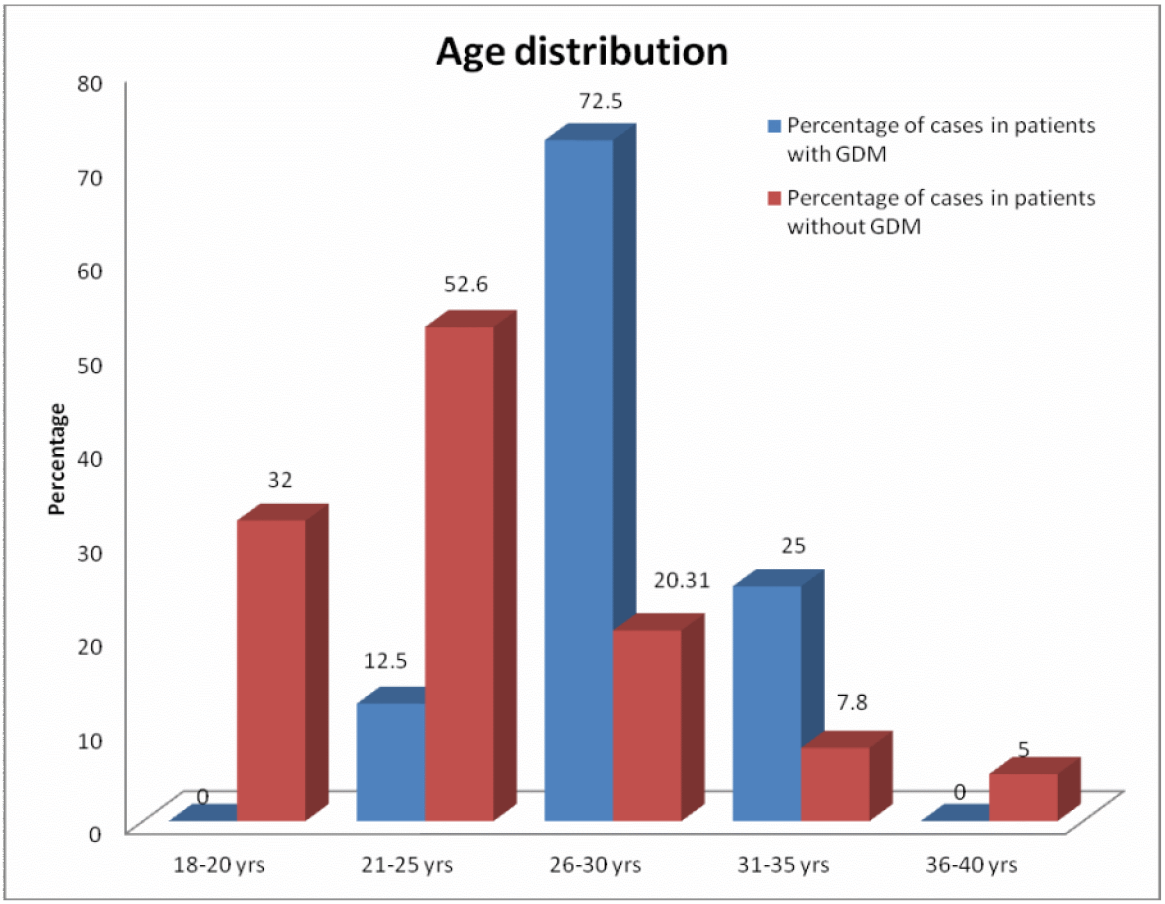

Age distribution
Percentage
80
70
60
50
40
30
20
10
0
Percentage of cases in patients with GDM
Percentage of cases in patients without GDM
0
32
12.5
52.6
72.5
20.31
25
7.8
0
5
18-20 yrs
21-25 yrs
26-30 yrs
31-35 yrs
36-40 yrs

Table 3

Socio Economic Status

Socio Economic Status		**Screening Positive for GDM**	**Screening Negative for GDM**	**Total**
Class I	No. of Pts			
	Percentage			
Class II	No. of Pts			
	Percentage			
Class III	No. of Pts	1	12	13
	Percentage	12.5%	6.25%	
Class IV	No. of Pts	3	56	59
	Percentage	37.5%	29.16%	
Class V	No. of Pts	4	124	128
	Percentage	50%	64.58%	

Chi Square value : 0.012 P Value : 0.912 NS

Majority of GDM patients belonged to Class v-50%,

Majority of GDM and non GDM patients belonged to non consanguineous marital status.

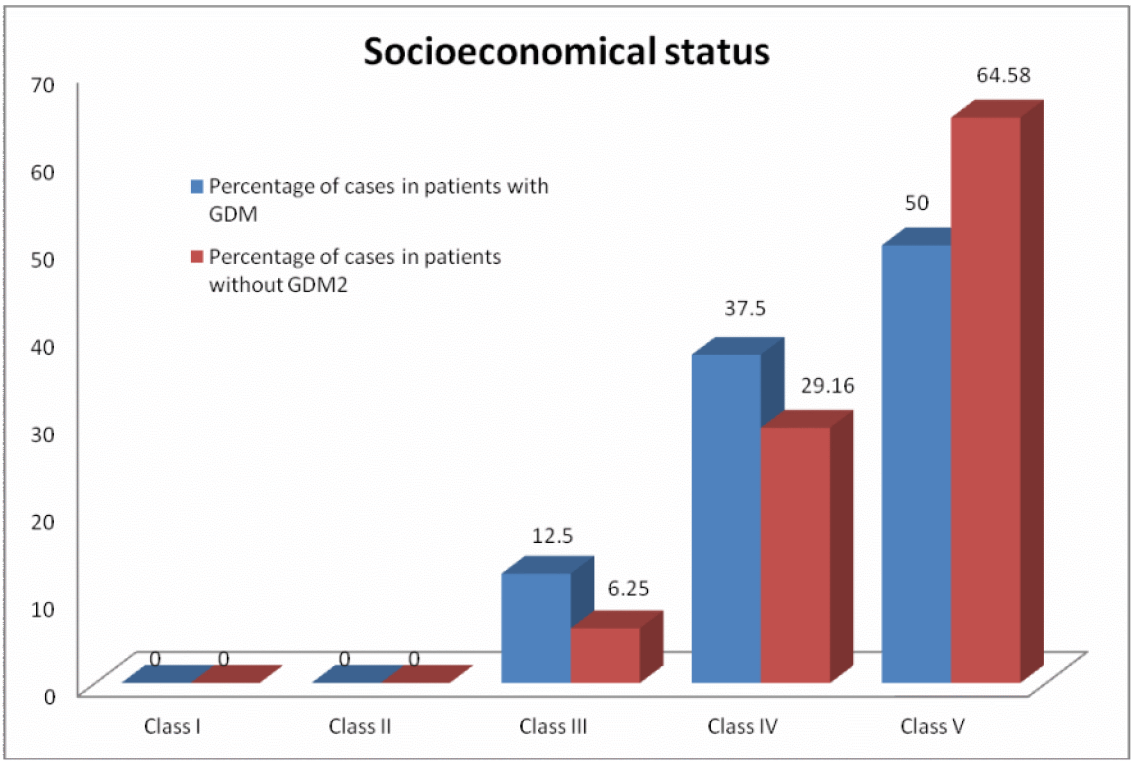

Socioeconomical status
70
60
50
40
30
20
10
0
Percentage of cases in patients with GDM
Percentage of cases in patients without GDM2
0
0
0
0
12.5
6.25
37.5
29.16
50
64.58
Class I
Class II
Class III
Class IV
Class V

Table 4

Gravida Status

Gravida Status		**Screening Positive for GDM**	**Screening Negative for GDM**	**Total**
Primi	No. of Pts	2	76	
	Percentage	25%	39.8%	
G2	No. of Pts	5	92	
	Percentage	62.5%	47.91%	
G3	No. of Pts	1	20	
	Percentage	12.5%	10.41%	
G4 and above	No. of Pts	Nil	4	
	Percentage		2.08%	

Chi Square value : 5.273 P Value : 0.004 Significant

- ❖ GDM was more common in multigravidas than primi ..

Among GDM patients 25% were primi.

62.5% among GDM were G2.12.5% were G3.

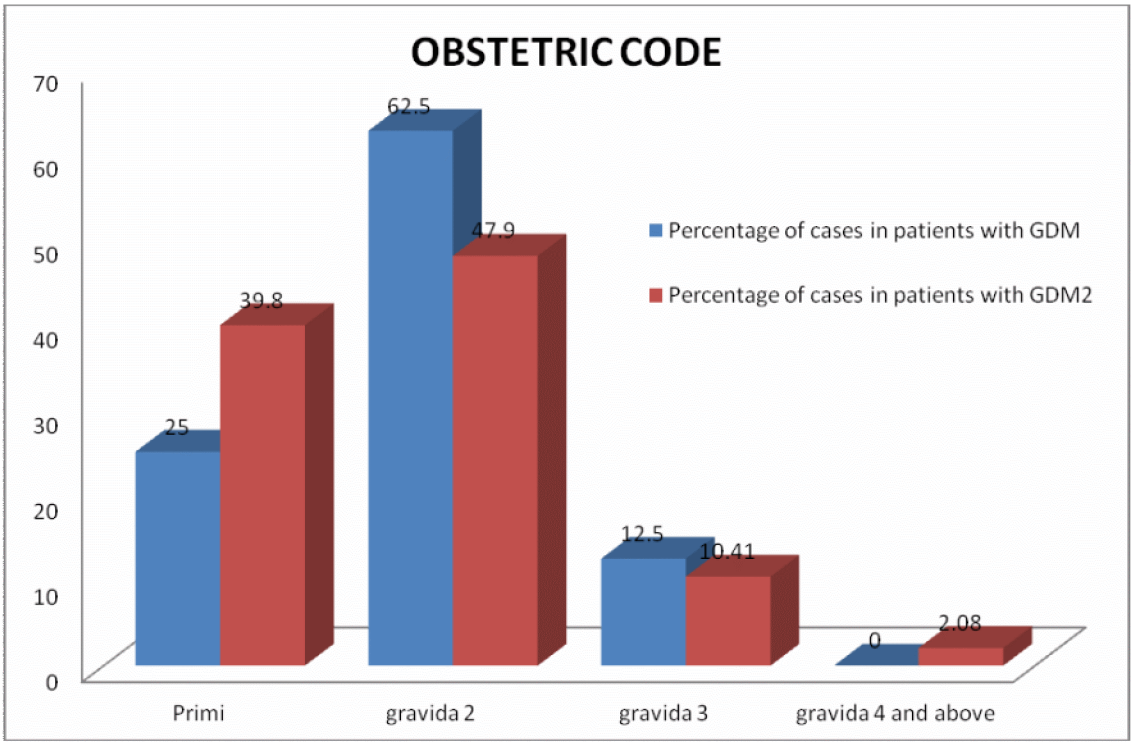

OBSTETRIC CODE
70
60
50
40
30
20
10
0
62.5
47.9
39.8
25
12.5
10.41
0
2.08
Percentage of cases in patients with GDM
Percentage of cases in patients with GDM2
Primi
gravida 2
gravida 3
gravida 4 and above

Table 5

Body Mass Index

BMI (in kg/m^2)		**Screening Positive for GDM**	**Screening Negative for GDM**	**Total**
Less than 18.5	No. of Pts	Nil	18	18
	Percentage		9.37%	
18.5-24.9	No. of Pts	2	132	134
	Percentage	25%	68.75%	
25-29.9	No. of Pts	5	36	41
	Percentage	62.5%	18.75%	
30 and above		1	6	7
		12.5%	3.12%	

Chi Square value : 6.78 P Value : 0.009 Significant

GDM more common in over weight and obese patient.

Among women with GDM 25% had BMI- 18.5-24.9.

Among women with GDM 62.5% had BMI 25-29.9.

Among women with GDM 12.5% had BMI above 30.

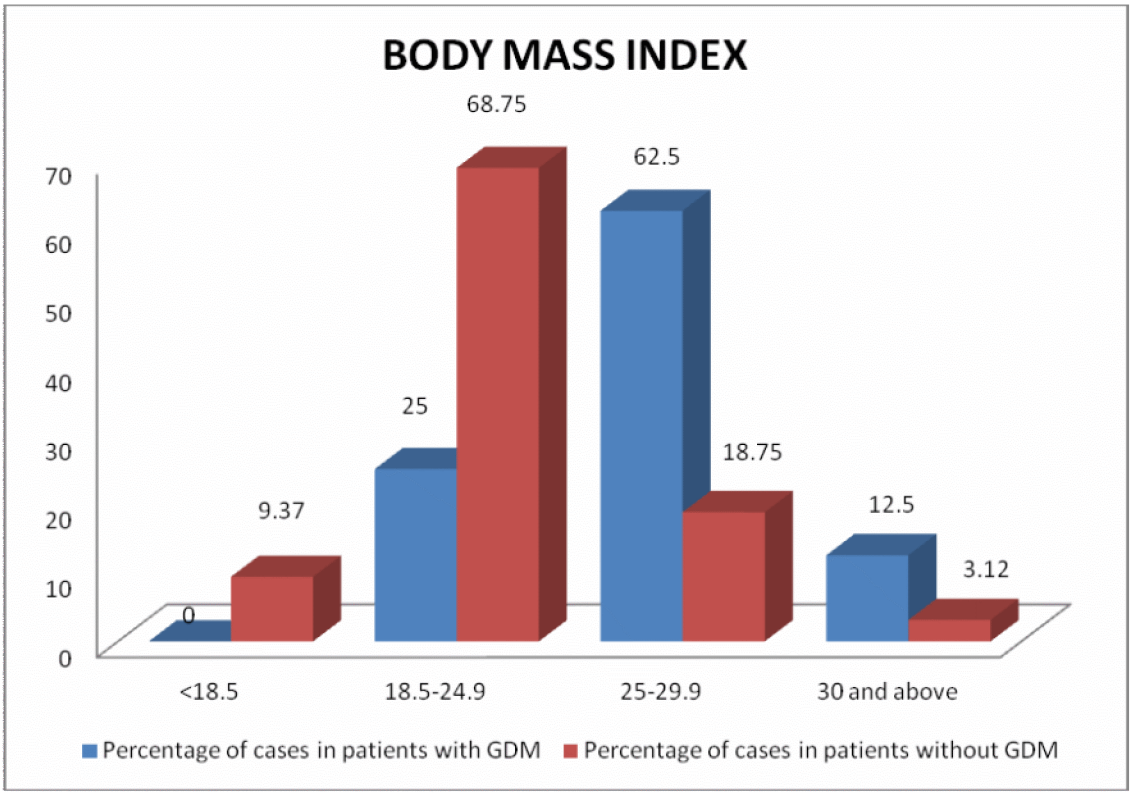

BODY MASS INDEX
68.75
62.5
25
18.75
12.5
9.37
3.12
0
70
60
50
40
30
20
10
0
<18.5
18.5-24.9
25-29.9
30 and above
Percentage of cases in patients with GDM
Percentage of cases in patients without GDM

Table 6

Family History of Diabetes Mellitus

Family History		**Screening Positive for GDM**	**Screening Negative for GDM**	**Total**
No Family history	No. of Pts			
	Percentage			
Father Diabetic	No. of Pts	2	22	
	Percentage	25%	11.45%	
Mother Diabetic	No. of Pts	1	16	
	Percentage	12.5%	8.33%	
No. of pts with family H/0. DM	No. of Pts	Nil	4	
	Percentage		2.08%	

Among GDM 37.5% had family history of Diabetes.

Chi Square value : 5.856 P Value : 0.012 Significant

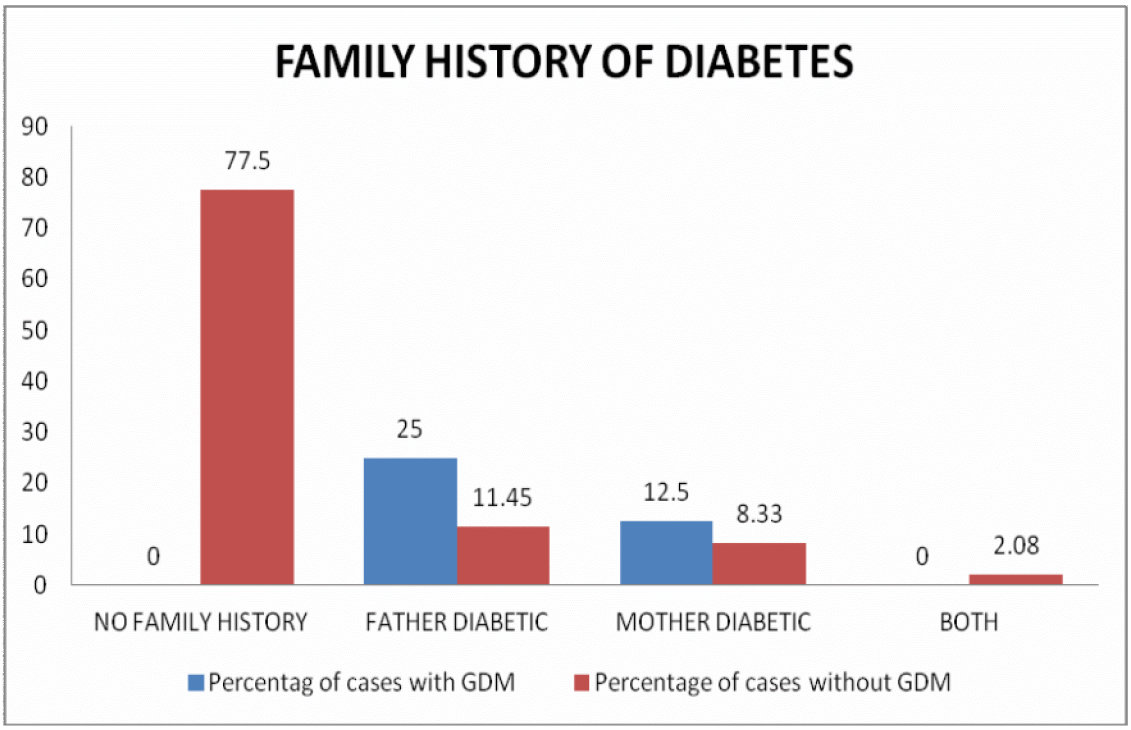

FAMILY HISTORY OF DIABETES
90
80
70
60
50
40
30
20
10
0
77.5
25
11.45
12.5
8.33
0
0
2.08
NO FAMILY HISTORY
FATHER DIABETIC
MOTHER DIABETIC
BOTH
Percentag of cases with GDM
Percentage of cases without GDM

Table 7

Risk Factors in the Past Pregnancy

Risk Factors		**Screening Positive for GDM**	**Screening Negative for GDM**	**Total**
History of Spontaneous Abortion	No. of Pts	1	8	10
	Percentage	12.5%	4.16%	
History of Delivery of baby with macrosomia	No. of Pts		1	1
	Percentage		0.52%	
History of Sudden IUD	No. of Pts	1	2	3
	Percentage	12.5%	1.04%	
History of Preterm Delivery	No. of Pts		4	4
	Percentage		2.08%	
History of unexplained neonatal death	No. of Pts		1	1
	Percentage		5.5%	
History of previous GDM with IGT	No. of Pts	1	1	2
	Percentage	12.5%	0.52%	
History of previous Pre-eclampsia	No. of Pts	1	6	7
	Percentage	12.5%	3.12%	
History of previous babies with congenital anamalies	No. of Pts	1	1	2
	Percentage	12.5%	0.52%	

Among GDM patient 1-12.5% had previous history of spontaneous abortion,1 had a baby with congenital anomaly,1-12.5% had previous history of previous GDM,History of IUD was present in 1 Patient.

Chi Square value : 6.842 P Value : 0.001 Significant

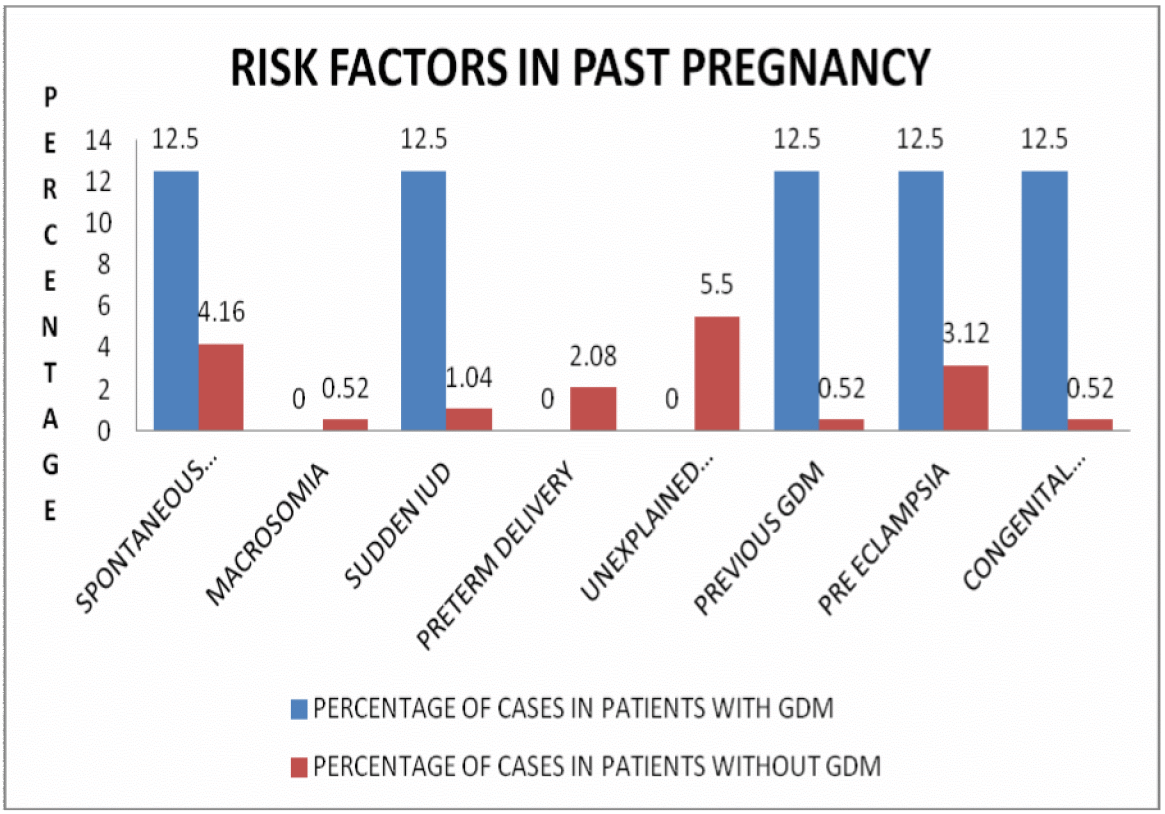

RISK FACTORS IN PAST PREGNANCY
PERCENTAGE
14
12
10
8
6
4
2
0
12.5
4.16
0
0.52
12.5
1.04
0
2.08
0
5.5
12.5
0.52
12.5
3.12
12.5
0.52
SPONTANEOUS...
MACROSOMIA
SUDDEN IUD
PRETERM DELIVERY
UNEXPLAINED...
PREVIOUS GDM
PRE ECLAMPSIA
CONGENITAL...
PERCENTAGE OF CASES IN PATIENTS WITH GDM
PERCENTAGE OF CASES IN PATIENTS WITHOUT GDM

Table 8

Risk Factors in Present Pregnancy

Risk Factors		**Screening Positive for GDM**	**Screening Negative for GDM**	**Total**
Obesity	No. of Pts	1	6	7
	Percentage	12.5%	3.12%	
	No. of Pts			
	Percentage			
	No. of Pts			
	Percentage			
Pre-eclampsia	No. of Pts	2	22	24
	Percentage	25%	11.45%	
Hydramnios	No. of Pts	1	3	4
	Percentage	12.25%	1.56%	
Congenital Malformations	No. of Pts		1	3
	Percentage		0.4%	
Macrosoima	No. of Pts	1	1	2
	Percentage	12.25%	0.4%	
IUD	No. of Pts		2	
	Percentage		1.04%	

Chi Square value : 7.842 P Value : < 0.001 Significant

Among GDM patients1(12.5%) had obesity.

Among GDM patient 2(25%) had Preeclampsia.

Among GDM patient had 1(12.5%)Macrosomia

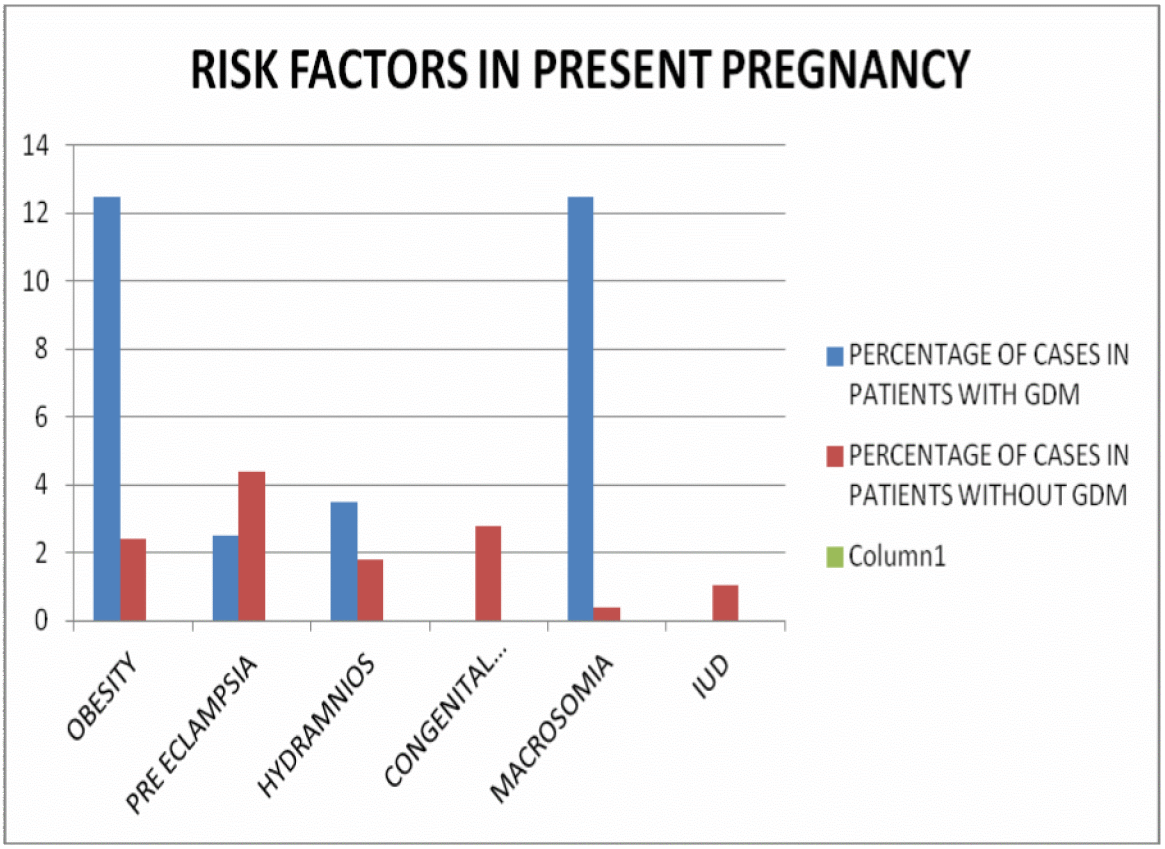

RISK FACTORS IN PRESENT PREGNANCY
14
12
10
8
6
4
2
0
OBESITY
PRE ECLAMPSIA
HYDRAMNIOS
CONGENITAL...
MACROSOMIA
IUD
PERCENTAGE OF CASES IN PATIENTS WITH GDM
PERCENTAGE OF CASES IN PATIENTS WITHOUT GDM
Column1

Table 9

Distribution of Blood Glucose Values in 2hrs 75 gm GTT

GTT	Number	Percentage
61-70	41	20.5%
71-80	36	18%
81-90	42	21%
91-100	32	16%
101-110	17	8.5%
111-120	12	6%
121-130	7	3.5%
131-140	2	2%
141-150	4	1.5%
151-160	4	2%
161-170	2	1%
171-180		
181-190		

Out of 200 12 had positive GCT report.

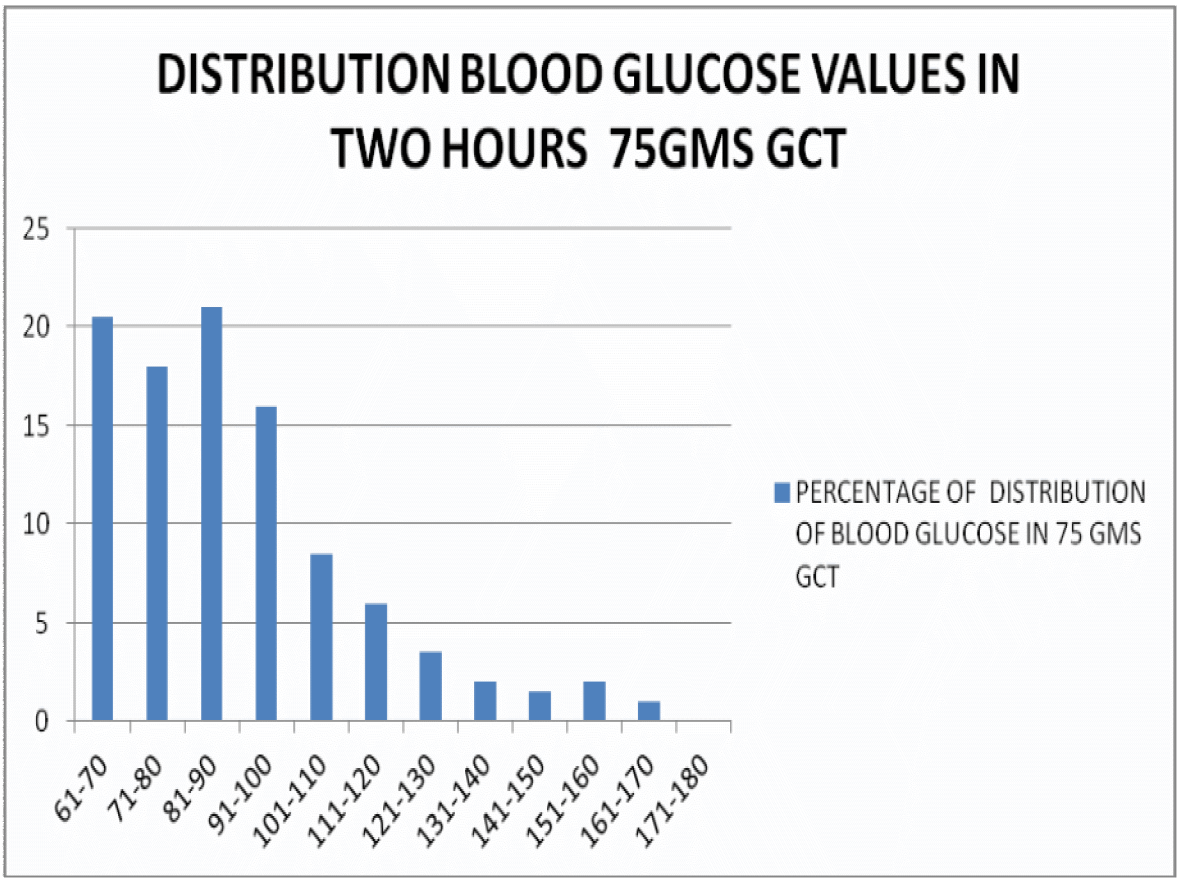

DISTRIBUTION BLOOD GLUCOSE VALUES IN TWO HOURS 75GMS GCT
25
20
15
10
5
0
61-70
71-80
81-90
91-100
101-110
111-120
121-130
131-140
141-150
151-160
161-170
171-180
PERCENTAGE OF DISTRIBUTION OF BLOOD GLUCOSE IN 75 GMS GCT

TABLE - 10

75 GRAMS OGTT VALUES (IADPSG CRITERIA)

Sl.No	Fasting	1 hour	2 hours
1	**106**	174	**155**
2	**96**	**196**	**157**
3	**102**	170	**156**
4	**108**	**186**	148
5	**96**	176	**158**
6	**104**	169	**158**
7	**98**	173	**162**
8	**109**	178	**162**
9	86	164	142
10	84	136	122
11	90	143	137
12	82	138	116

Out of 12, 8 patients are positive for GDM using IADPSG criteria.

TABLE - 11

HbA1C values in GDM cases

Sl.No	HbA1C values
1	**4.6**
2	**4.8**
3	**4.4**
4	**4.5**
5	**5.2**
6	**4.1**
7	**4.4**
8	**4.7**

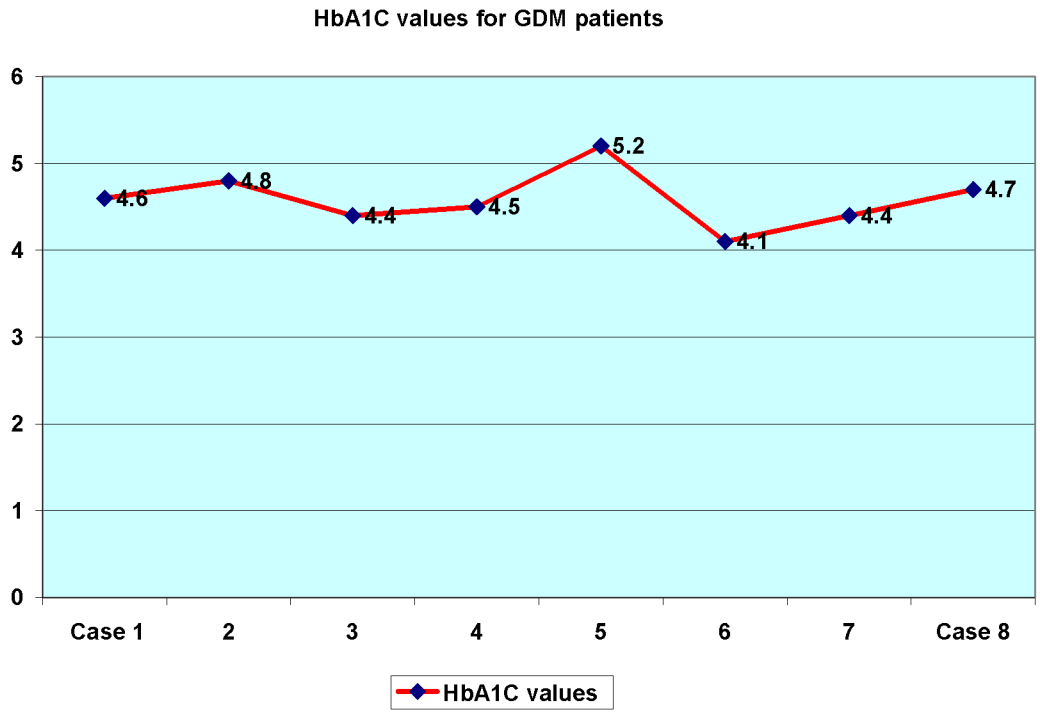


Table 12

In the Intrapartum Period

I.Timing of Delivery

Gestational age in weeks at delivery		**Screening Positive for GDM**	**Screening Negative for GDM**
< 37 weeks	No. of Pts	2	24
	Percentage	25%	12.5%
➢ 37 weeks	No. of Pts	6	168
	Percentage	75%	87.5%

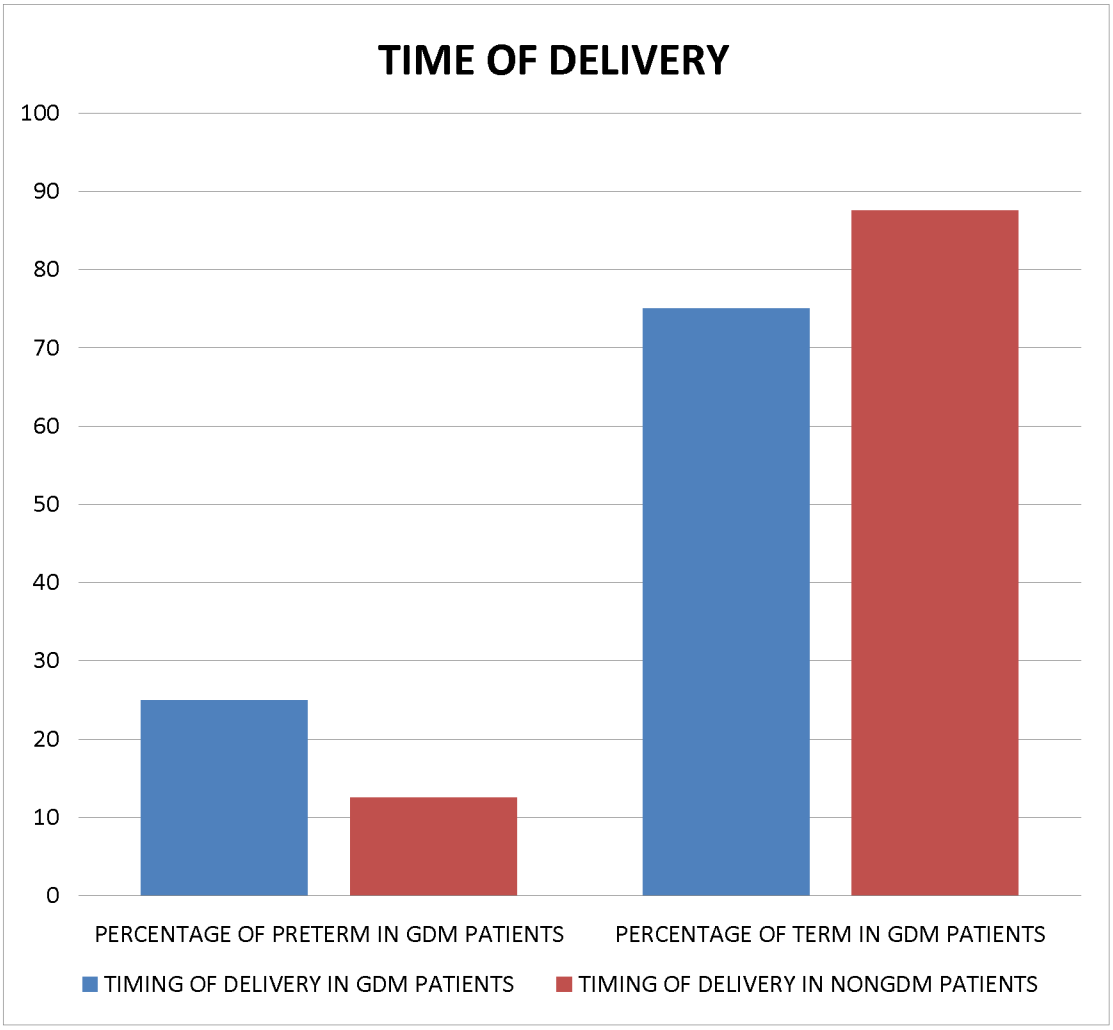

TIME OF DELIVERY
100
90
80
70
60
50
40
30
20
10
0
PERCENTAGE OF PRETERM IN GDM PATIENTS
PERCENTAGE OF TERM IN GDM PATIENTS
TIMING OF DELIVERY IN GDM PATIENTS
TIMING OF DELIVERY IN NONGDM PATIENTS

Table 13

II. Mode of Delivery

Mode		**Screening Positive for GDM**	**Screening Negative for GDM**
Labour Natural	No. of Pts		26
	Percentage		13.54%
Labour Natural with episiotomy	No. of Pts	2	106
	Percentage	25%	55.2%
Outlet Forceps	No. of Pts		8
	Percentage		4.1%
Assisted breech	No. of Pts		2
	Percentage		1.04%
Emergency Section	No. of Pts	2	32
	Percentage	25%	16.66%
Elective Section	No. of Pts	4	18
	Percentage	50%	9.37%

In our study cesarean section in GDM patients was about 75%.

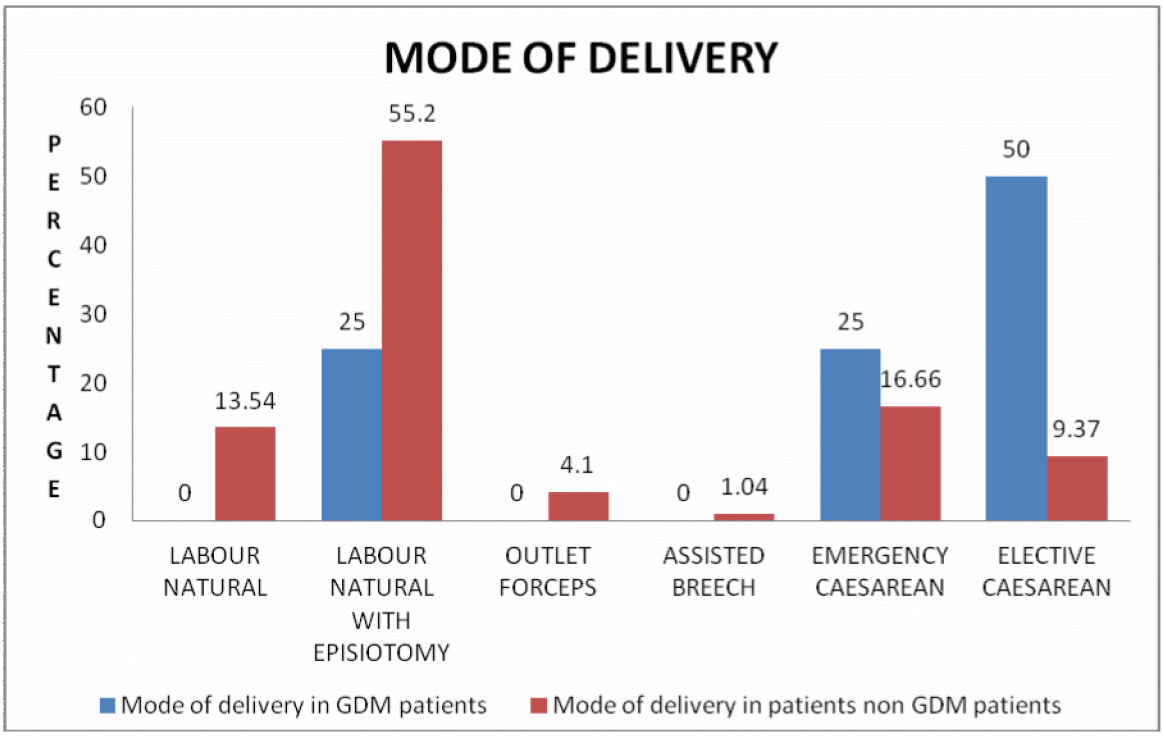

MODE OF DELIVERY
PERCENTAGE
60
50
40
30
20
10
0
0
13.54
25
55.2
0
4.1
0
1.04
25
16.66
50
9.37
LABOUR NATURAL
LABOUR NATURAL WITH EPISIOTOMY
OUTLET FORCEPS
ASSISTED BREECH
EMERGENCY CAESAREAN
ELECTIVE CAESAREAN
Mode of delivery in GDM patients
Mode of delivery in patients non GDM patients

TABLE - 14

MODE OF DELIVERY AMONG GDM PATIENTS

Labour natural with episiotomy	-	2
Lscs	-	6
Elective Lscs	-	4
Emergency Lscs	-	2

INDICATION FOR LSCS IN GDM PATIENTS

Primi with CPD 1st degree with GDM

Primi with CPD 1st degree with GDM with mild PIH

Fetal distress

Previous LSCS-1.

Fetal alarm signal-1.

Previous LSCS with PROM-1

Table 15

Management of GDM Patients

Management		**Screening Positive for GDM**
Insulin	No. of Pts	7
	Percentage	87.5%
Diet Alone	No. of Pts	1
	Percentage	12.5%

Initially all patients were treated with meal plan and patients were started on insulin due to poor glycemic control. 1 patient is treated with meal plan alone till delivery.

Among GDM Patients 12.5% treated with meal plan only.

Among GDM Patients 87.5% treated with insulin therapy.

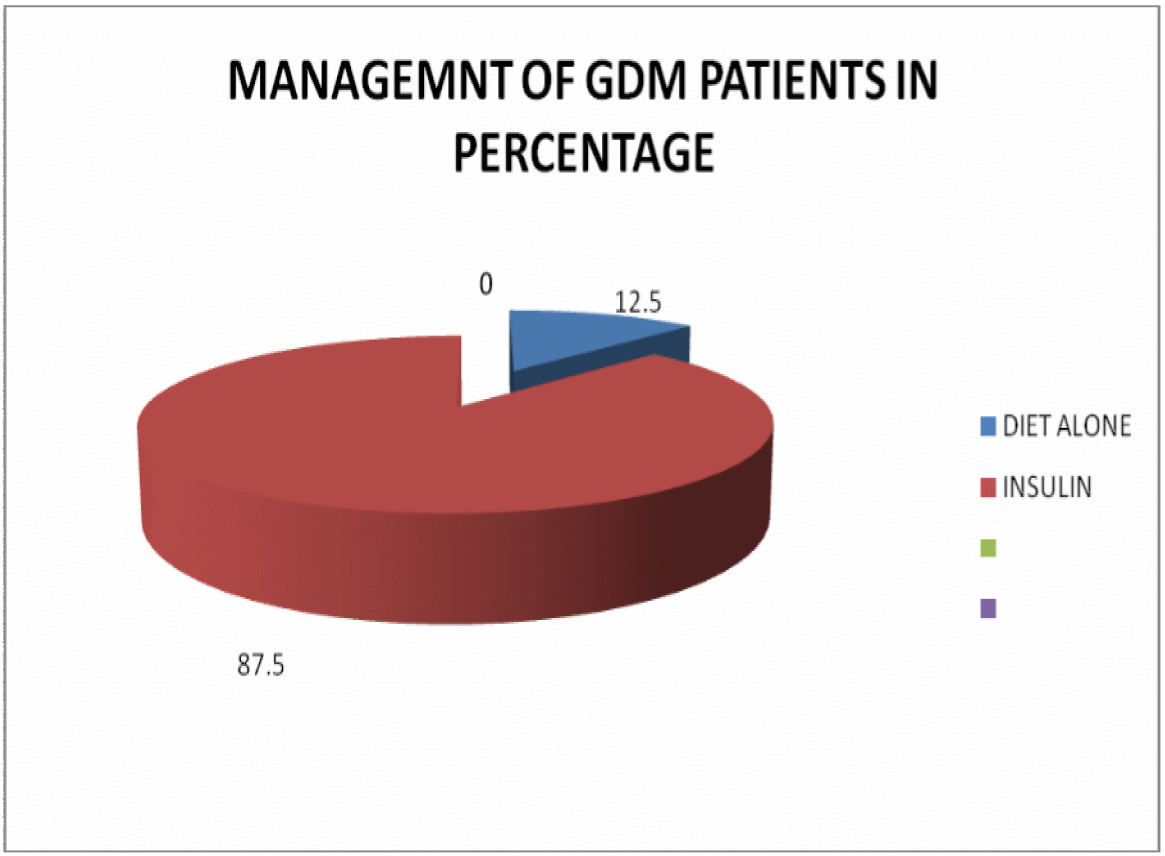

MANAGEMNT OF GDM PATIENTS IN PERCENTAGE
0
12.5
87.5
DIET ALONE
INSULIN

Table 16

Fetal Outcome

1.Apgar score in babies at 5 mins

Apgar Group		**Screening Positive for GDM**
8 and above	No. of Pts	6
	Percentage	75%
5-7	No. of Pts	2
	Percentage	25%
1-4	No. of Pts	
	Percentage	

Majority of infants born to GDM patients had good Apgar score.

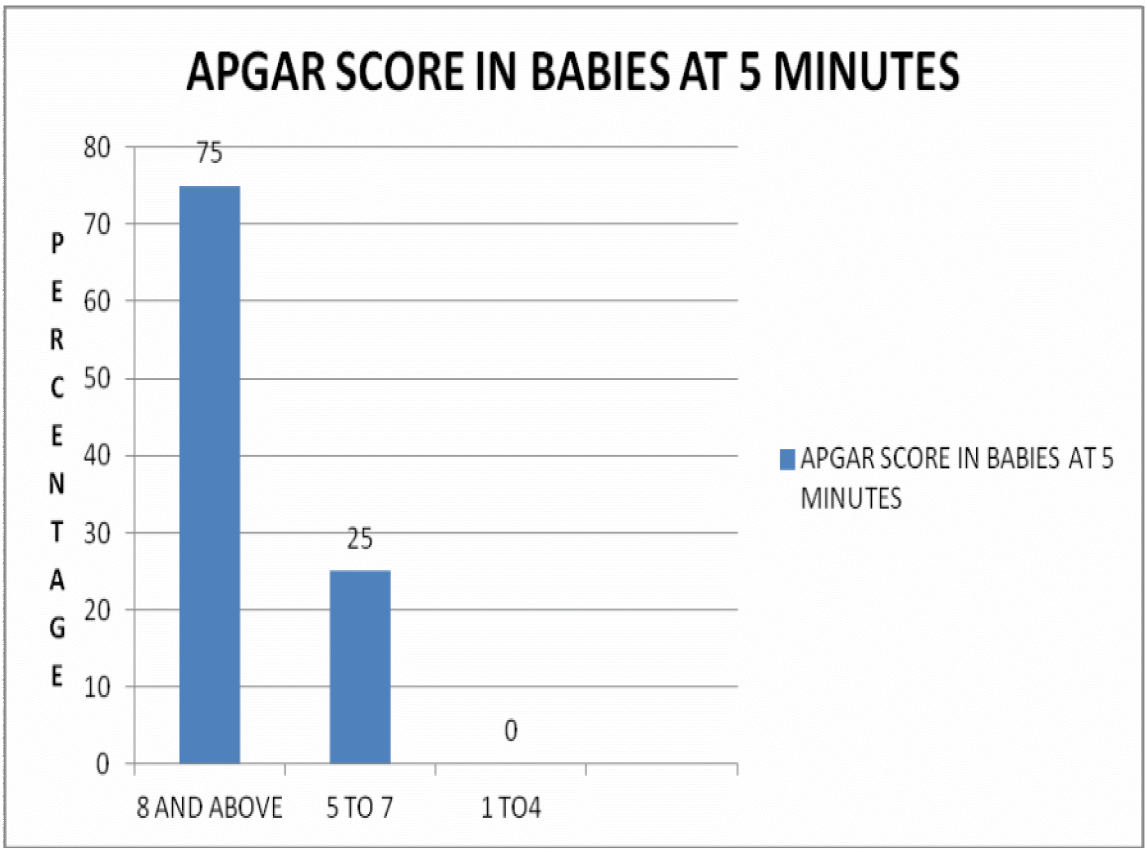

APGAR SCORE IN BABIES AT 5 MINUTES
PERCENTAGE
80
70
60
50
40
30
20
10
0
75
25
0
8 AND ABOVE
5 TO 7
1 TO4
APGAR SCORE IN BABIES AT 5 MINUTES

Table 17

2. Birth Weight

Weight		**Screening Positive for GDM**	**Screening Negative for GDM**
Less than 2 kg	No. of Pts		12
	Percentage		6.25%
2.1 to 2.4 kg	No. of Pts	1	16
	Percentage	12.5%	8.33%
2.5 to 2.9 kg	No. of Pts	3	133
	Percentage	37.5%	69.27%
3-3.4 kg	No. of Pts	2	22
	Percentage	25%	11.45%
3.5 to 3.9 kg	No. of Pts	1	8
	Percentage	12.5%	4.16%
4 and above	No. of Pts	1	1
	Percentage	12.5%	0.52%

12.5% of GDM had babies between 2.1-2.4kg.

37.5% of GDM had babies between 2.5-2.9kg.

25% of GDM had babies between 3-3.4kg.

12.5% of GDM had babies between 3.5-3.9kg.

12.5% of GDM had babies above 4 kg.

BIRTH WEIGHT

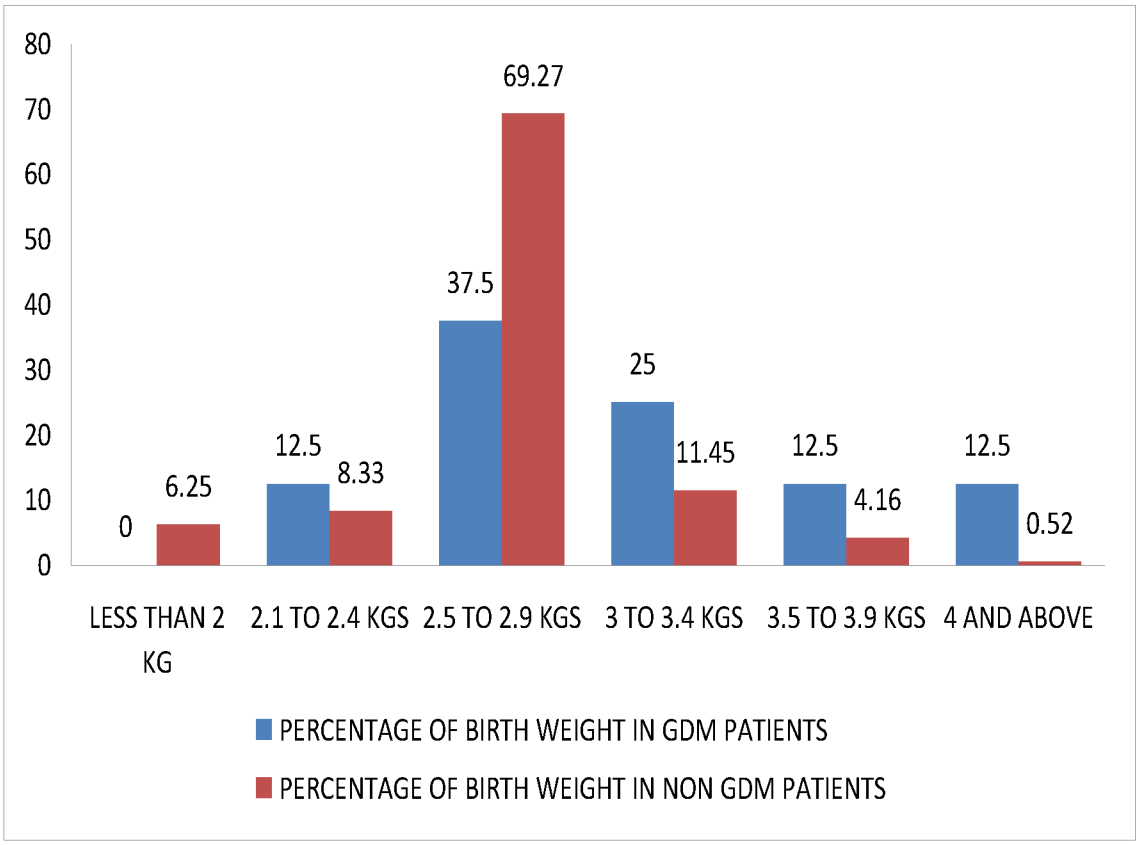


Table 18

2. Neonatal Complications

Complications		Screening Positive for GDM	Screening Negative for GDM
Hypoglycemia	No. of Pts	1	0
	Percentage	12.5%	
Hypocalcemia	No. of Pts		
	Percentage		
Hypomagnesemia	No. of Pts		
	Percentage		
Hyperbilirubinemia	No. of Pts	1	11
	Percentage	12.5%	5.72%
Congenital anomalies	No. of Pts		3
	Percentage		1.56%
RDS	No. of Pts		4
	Percentage		2.08%
TTN	No. of Pts		5
	Percentage		3.08%

Among GDM PATIENTS 12.5% had hypoglycemia.

Among GDM Patients 12.5% had hyperbilirubinemia.

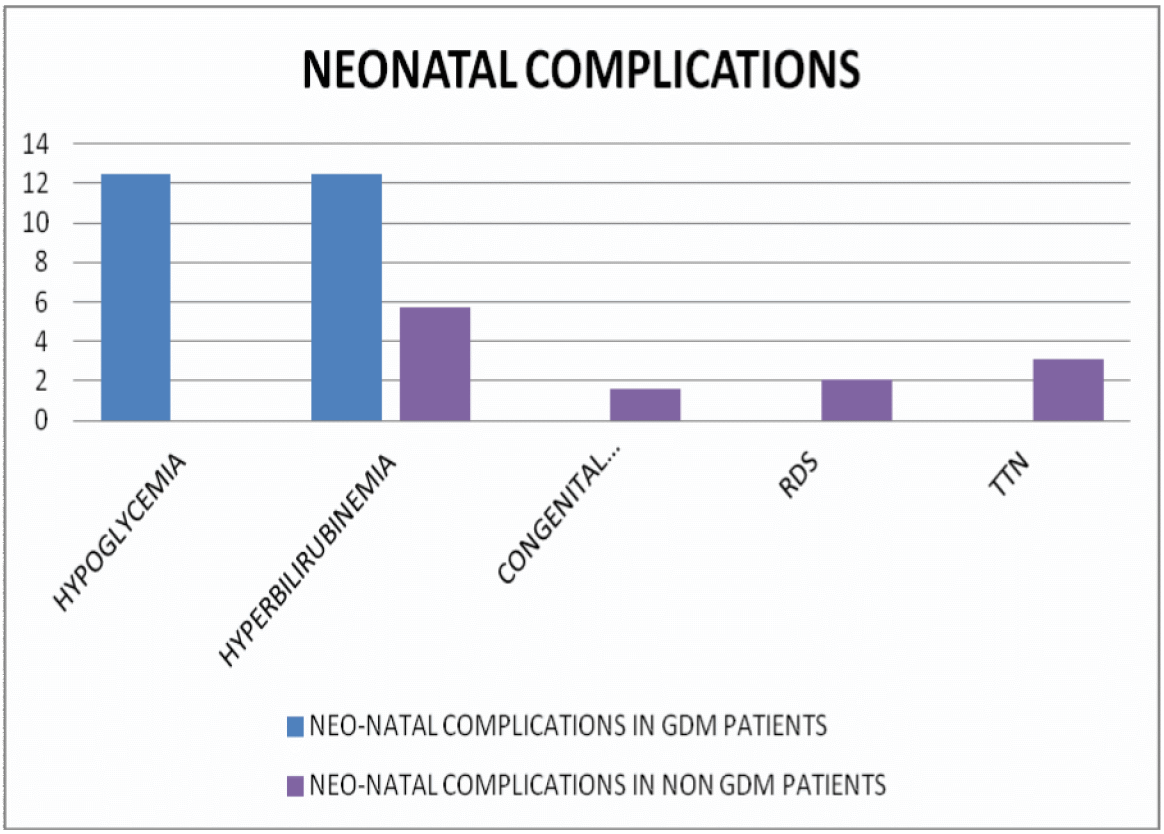
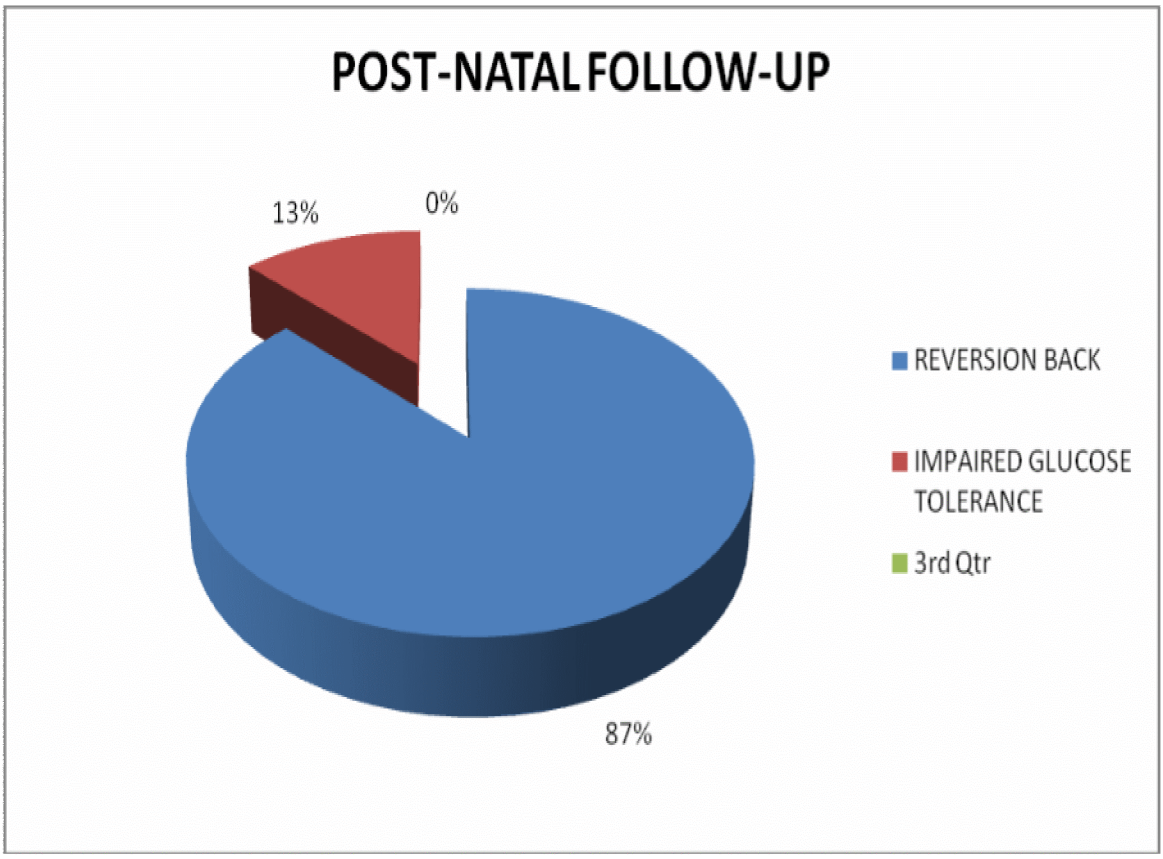

NEONATAL COMPLICATIONS
14
12
10
8
6
4
2
0
HYPOGLYCEMIA
HYPERBILIRUBINEMIA
CONGENITAL...
RDS
TTN
NEO-NATAL COMPLICATIONS IN GDM PATIENTS
NEO-NATAL COMPLICATIONS IN NON GDM PATIENTS

Table 19

Postnatal Follow Up Using 2 hrs 75gm OGTT

Postnatal	Screening Positive for GDM
Reversion Back	**7**
Impaired glucose tolerance	**1**

Six weeks after delivery 8 patients turned for follow up and they were subjected to 75 gram OGTT. 7 reverted back to normal and 1 had impaired glucose tolerance.


POST-NATAL FOLLOW-UP
13%
0%
87%
REVERSION BACK
IMPAIRED GLUCOSE TOLERANCE
3rd Qtr

DISCUSSION

Gestational diabetes mellitus is one of the commonest complications of pregnancy. Gestational Diabetes Mellitus has been associated with increased risk for complications of pregnancy but also longterm risk for both mother and fetus. GDM in the mother is associated with increased risk of overt diabetes later in life. A higher risk for developing metabolic and cardiovascular disease has been reported for women who develop GDM during pregnancy.

Gestational diabetes Mellitus is a risk factor for both mother and fetus. This risk increases proportionally to the maternal blood Sugar concentration. So, we have to screen all the antenatal patients with screening test, to reduce perinatal morbidity and mortality and maternal morbidity.

Indians are ethnically more prone to develop diabetes. So, universal screening should be done in Indian population.

In this study, we used 75 gm GCT as a screening test, and 75 gms **IADPSG OGTT** as a diagnostic test. HbA1C was done to patients positive for GDM.

In this study, incidence of GDM is 4% by IADPSG criteria. The prevalence of GDM in India varies from 3.8 to 21% depending on the

geographical location and ethnicity. Overall, 1 to 5% of prevalence was reported by Isaac and Hema Divakar 2011.[36] Incidence of GDM in our Govt. Rajaji Hospital by ADA criteria is 3.6%.

Age Distribution

Most of the patients with GDM were in the age group of 26 to 30 years.

72.5%among GDM were between 26 to 30 years.

Advancing maternal age is associated with increased trend of gestational diabetes. It is similar to earlier study done by SESIAH ET AL 2008,[37] NHS 2006 DATA.

Body Mass Index

In our study 62.5% had Body Mass index between (25 – 29.9) .

12.5% had BMI above 30, 25% had BMI between 18.5-24.9

Which is statistically significant.

When the BMI is high there is a strong link between GDM & BMI

(BJOG 2011)

Increased BMI is associated with GDM(BIANCO ET AL 1998).

We noticed increased trend of prevalence of GDM as the age

and BMI increased which is similar to earlier study(Sesiah et al)[38]

FAMILY HISTORY OF DIABETES:

In our study Father was diabetic in 25% of GDM patients.

Mother was diabetic in 12.5% of diabetic patients

GDM was present in 37.5% of the patients with positive

Family history.

GDM was present in 11.6% with positive family history

GESTATIONAL HYPERTENSION

In our study, the incidence of Gestational hypertension among GDM patients was 25%.

YANG ET AL – 2006[39] reported the incidence of 28% of Gestational hypertension in GDM patients. In non-diabetic patients, the incidence is 9% which is similar to our study.

Suhonen and Torano in 1993[40] reported incidence of preeclampsia is two times more common in GDM patients.

Garner et al 1995[41] have shown that preeclampsia was twice high in GDM patients.

Hydramnios

In our study, the incidence of Hydramnios was 12.5% among GDM

Incidence of polyhydramnios in GDM is 10% in NCBI study which is similar to our study.

Preterm labour

In our study, the incidence of preterm labour was 25% which is similar to a Canadian study by YANG ET AL 2006.[42] Joslin diabetes center incidence is 26%. YANH ET AL 2006. Incidence of preterm birth among GDM patients. 9 percent of GDM women spontaneously delivered before 35 weeks compared with 4.5 percent of non-diabetic woman. 7% of diabetic women underwent indicated preterm delivery. Single most important factor for preterm birth in GDM is preeclampsia.

MODE OF DELIVERY

In our study of 8 GDM patients , 2 delivered by Labour natural with episiotomy, that is 25%. 6 delivered by caesarian section. 4(50%) had elective caesarian section. 2(25%) had emergency caesarian section.

HAWTHORNE G ROBSON ET AL[43] – found the rate of caesarian section is 75% in diabetic patient.

GABBE ET AL[44] – Caesarian section ranges from 50 to 80% among GDM

Rate of caesarian section in overt diabetes is 80 percent in Parkland Hospital.

In our study the incidence is 75%

In our study, the incidence of macrosomia is 12.5%

In our study 12.5% GDM patients, had babies weighing between 2 – 2.4 kg. 37.5% had babies weighing between 2 – 2.4 kg. 37.5% had babies weighing between 2.5 to 2.9 kg.

25% had babies weighing between 3 – 3.4 kg

12.5% had babies weighing between 3.5 to 3.9 kg. Incidence of macrosomia is reduced due to the treatment given to GDM patients.

Fetal macrosomia complicates 50% of pregnancies in women with GDM-

(GABBE)[45]

LANDON and Colleagues[46] using daily capillary glucose values, reported a rate of 9% macrosomia when mean values were below 110 mg/dl

Compared with 34% when less optimal control was achieved.

HYPOGLYCEMIA

With near physiological control of maternal glucose levels during pregnancy. Overall rates of Hypoglycemia is 5 to 15% (Steven

G.GABBE)[47] but in Uncontrolled diabetes , the incidence is 50%.In our study the incidence is 12.5%.

RESPIRATORY DISTRESS SYNDROME

Women with well controlled diabetes the risk of respiratory distress syndrome was no higher than that observed in general population.(Steven G. GABBE)

Kjos and Walther[48] reported the incidence of 0.95%.

In our study there is no respiratory symptom.

HYPERBILIRUBINEMIA:

Hyperbilirubinemia has been reported in as many as 25% to 53% of pregnancies complicated by pregestational and 38% of pregnancies in women with GDM. In our study the incidence is 12.5%.

SUMMARY

1. Incidence of Gestational in 200 selected population 4%
2. HbA1C levels are below 5.1% in GDM cases
3. Majority of GDM patients were in the age of 26-30 yrs [72.5%]
4. 75% GDM patients were multigravida
5. 62.5% of GDM patients had BMI more than 25 kg/m^2

   12.5 % of GDM patients had BMI more than 30 kg/m^2
6. 37.5 % of patients with GDM had family history of diabetes
7. Among GDM patients 25% had preeclampsia
8. Among GDM patients 12.5% had polyhydramnios
9. Among GDM patients 12.5% had macrosomia
10. Among GDM patients 25% had labour natural with episiotomy and 75% had cesarean section.
11. Among GDM patients 12.5% were treated with meal plan .87.5% were treated with insulin therapy.
12. Among GDM patients 12.5% had hypoglycemia,12.5% had hyperbilirubinemia.

CONCLUSION

- Increase in the prevalence of diabetes worldwide by 40% in the past 10 years. Prevalence in 1990 4.9 percent, [Narayan et al][49] 1999 6.9 percent.
- This increased prevalence is due to obesity and life style changes such as decreased exercise. Increase in the prevalence of type II DM lead to an increasing number of pregnancies with complications.
- GDM patients are definitely associated with significantly increased maternal and perinatal morbidity. All complications associated with GDM are potentially preventable with early recognition, close monitoring and proper treatment.
- Early imprinting in inutero life of the fetus can have effects later in life. Fetal exposure of diabetes leading to childhood obesity, glucose intolerance, diabetes in adult life.

  More than Half of the women with gestational diabetes in the ensuing 20 yrs.
- There is high prevalence of diabetes mellitus and its early onset among Indians and its complications during pregnancy and its long term complications such as maternal diabetes mellitus and diabetes in offspring necessitate the importance of early screening and treatment.

- ❖ Early screening identified undiagnosed type II DM. Overt diabetes has more complications like congenital malformation, diabetic nephropathy, neuropathy, Diabeticketoacidosis.
- ❖ Hence an appropriate method of screening has been much emphasized.
- ❖ Early diagnosis of GDM reduces maternal and perinatal mortality and morbidity.

Incidence of GDM in Government Rajaji Hospital using ADA criteria is 3.6%

Out of 200 patients taken up for study, Incidence of GDM in our study is 4%. Selective screening is applicable for women belonging to ethnic group with the low prevalence of GDM.

Whereas ethnically, Indian women are more prone to develop glucose intolerance.

Indians have eleven fold increased risk. Compared to whites, necessitating Universal screening during pregnancy. In our study, only 50% GDM patients belong to patients indicated for screening as per ADA. If we followed selective screening, we could have been missed 50% of GDM cases in selective screening.

In our study, 200 patients were screened by Glucose challenge test using 75 gm of glucose irrespective of previous meal, GCT measured after

2 hours. OGTT done by IADPSG Criteria. HbA1C was done to GDM cases diagnosed were treated. Prophylactic steroids given to GDM patients.

Hyperglycemia and adverse pregnancy outcomes (HAPO) study specifically evaluated the effect of maternal hyperglycemia, less severe than overt diabetes, on pregnancy outcomes. It demonstrated that there is a continuous relationship between maternal hyperglycemia and Pregnancy outcomes. IADPSG is based on HAPO trial.

2011 ADA Endorsed IADPSG criteria. Early diagnosis of GDM reduces maternal and perinatal mortality and morbidity.

During our study, detected GDM patients were closely monitored and treated with either insulin or meal plan which reduce the adverse obstetric and perinatal outcome.

BIBLIOGRAPHY

1. Narayan KM, Boyle JP, Thompson TJ, et al: Lifetime risk for diabetes mellitus in the United States. JAMA 290:1884, 2003 [PMID: 14532317.
2. Coustan DR et al, Summary and recommendations of the Fifth International Workshop-Conference on Gestational Diabetes Mellitus,Diabetes Care. 2007 Jul;30 Suppl 2:S251-60. doi: 10.2337/dc07-s225.
3. Seshiah V[1], Balaji V, Balaji MS, Paneerselvam A, Arthi T, Thamizharasi M, Datta M Prevalence of gestational diabetes mellitus in South India (Tamil Nadu)--a community based studyJ Assoc Physicians India. 2008 May;56:329-33.
4. Gabbe S, Gregory R, Power M, et al: Management of diabetes mellitus by obstetricians-gynecologists. Obstet Gynecol 103:1229, 2004
5. Yang J, Cummings EA, O'Connell C, et al: Fetal and neonatal outcomes of diabetic pregnancies. Obstet Gynecol 108:644, 2006
6. Sibai BM, Caritis S, Hauth J, et al: Risks of preeclampsia and adverse neonatal outcomes among women with pregestational diabetes.mellitus. Am J Obstet Gynecol 182:364, 2000
7. Garner PR: Type 1 diabetes and pregnancy. Lancet 346:966, 1995
8. Pedersen J: The Pregnant Diabetic and Her Newborn, 2nd ed. Baltimore, Williams & Wilkins, 1977, p 211
9. Temple RC, Aldridge V, Stanley K, et al: Glycaemic control throughout pregnancy and risk of pre-eclampsia in women with type 1diabetes. BJOG 113:1329, 2006
10. Stamler EF, Cruz ML, Mimouni F, et al: High infectious morbidity in pregnant women with insulin-dependent diabetes: an understatedcomplication. Am J Obstet Gynecol 163:1217, 1990
11. Dashe JS, McIntire DD, Twickler DM: Effect of maternal obesity on the ultrasound detection of anomalous fetuses. Obstet Gynecol 113(5):1001, 2009
12. Sibai BM, Viteri OA: Diabetic ketoacidosis in pregnancy. Obstet Gynecol 123(1):167, 2014

13. Ferrara A: Increasing prevalence of gestational diabetes. Diabetes Care 30:S141, 2007
14. Feig DS, Palda VA: Type 2 diabetes in pregnancy: a growing concern. Lancet 359:1690, 2002
15. Getahun D, Fassett MJ, Jacobsen SJ: Gestational diabetes: risk of recurrence in subsequent pregnancies. Am J Obstet Gynecol 203:467,2010
16. Baraban E, McCoy L, Simon P: Increasing prevalence of gestational diabetes and pregnancy-related hypertension in Los Angeles
17. County, California, 1991–2003. Prev Chronic Dis 5:A77, 2008
18. Saudek CD: Progress and promise of diabetes research. JAMA 287:2582, 2002
19. Kim C, Cheng YJ, Beckles GL: Cardiovascular disease risk profiles in women with histories of gestational diabetes but without current diabetes. Obstet Gynecol 112(4):875, 2008
20. Landon MB, Mele L, Spong CY, et al: The relationship between maternal glycemia and perinatal outcome. Obstet Gynecol 117(2):218,2011
21. Dempsey JC, Sorensen TK, Williams MA, et al: Prospective study of gestational diabetes mellitus in relation to maternal recreational physical activity before and during pregnancy. Am J Epidemiol 159:663, 2004
22. Brankston GH, Mitchell BF, Ryan EA, et al: Resistance exercise decreases the need for insulin in overweight women with gestational diabetes mellitus. Am J Obstet Gynecol 190:188, 2004
23. Bung P, Bung C, Artal R, et al: Therapeutic exercise for insulin-requiring gestational diabetes: effects on the fetus—results of a randomized prospective longitudinal study. J Perinat Med 21:125, 1993
24. Langer O, Yogev Y, Xenakis EMJ, et al: Insulin and glyburide therapy: dosage, severity level of gestational diabetes, and pregnancy outcome. Am J Obstet Gynecol 192:134, 2005
25. Myla E Moretti MSc, Assistant Director, Motherisk Program, Hospital for Sick Children, Toronto, Ontario, Canada doi: 10.1345/aph.1K577Ann Pharmacother April 2008 vol. 42no. 4 483-490

26. Hebert MF, Naraharisetti SB, Krudys KM, et al: Are we optimizing gestational diabetes treatment with glyburide? The pharmacologic basis for better clinical practice. Clin Pharmacol Ther 85(6):607, 2009
27. Glueck CJ, Goldenberg N, Wang P, et al: Metformin during pregnancy reduces insulin, insulin resistance, insulin secretion, weight,testosterone and development of gestational diabetes: prospective longitudinal assessment of women with polycystic ovary syndromefrom preconception throughout pregnancy. Hum Reprod 19:510, 2004
28. Harborne L, Fleming R, Lyall H, et al: Descriptive review of the evidence for the use of metformin in polycystic ovary syndrome. Lancet361:1894, 2003
29. Rowan JA, Hague WM, Wanzhen G, et al: Metformin versus insulin for the treatment of gestational diabetes. N Engl J Med 358:2003,2008
30. Coustan DR et al, Summary and recommendations of the Fifth International Workshop-Conference on Gestational Diabetes Mellitus,Diabetes Care. 2007 Jul;30 Suppl 2:S251-60. doi: 10.2337/dc07-s225
31. Hawkins JS, Lo JY, Casey BM, et al: Diet-treated gestational diabetes: comparison of early versus routine diagnosis. Am J Obstet Gynecol, 198:287, 2008
32. Gabbe S, Gregory R, Power M, et al: Management of diabetes mellitus by obstetricians-gynecologists. Obstet Gynecol 103:1229, 2004
33. Conway DL, Gonzales O, Skiver D: Use of glyburide for the treatment of gestational diabetes: the San Antonio experience. J MaternFetal Neonatal Med 15:51, 2004
34. Holmes HJ, Casey BM, Lo JY, et al: Likelihood of diabetes recurrence in women with mild gestational diabetes (GDM). Am J Obstet Gynecol 189 (6):161, 2003
35. Pallardo F, Herranz L, Garcia-Ingelmo T, et al: Early postpartum metabolic assessment in women with prior gestational diabetes. Diabetes Care 22:1053, 1999 [PMID: 10388966

36. Coustan DR et al, Summary and recommendations of the Fifth International Workshop-Conference on Gestational Diabetes Mellitus,Diabetes Care. 2007 Jul;30 Suppl 2:S251-60. doi: 10.2337/dc07-s225
37. Hema Divakar, Isaac Manyonda, Battling with prevalence of GDM,10.5005 jp.journals-10016-1026
38. Seshiah V[1], Balaji V, Balaji MS, Paneerselvam A, Arthi T, Thamizharasi M, Datta M Prevalence of gestational diabetes mellitus in South India (Tamil Nadu)--a community based studyJ Assoc Physicians India. 2008 May;56:329-33.
39. Seshiah V[1], Balaji V, Balaji MS, Paneerselvam A, Arthi T, Thamizharasi M, Datta M Prevalence of gestational diabetes mellitus in South India (Tamil Nadu)--a community based studyJ Assoc Physicians India. 2008 May;56:329-33.
40. Yang J, Cummings EA, O'Connell C, et al: Fetal and neonatal outcomes of diabetic pregnancies. Obstet Gynecol 108:644, 2006
41. Lauri Suhonen,Helsinki Glycemic Control in Diabetic Pregnancies:Effects on Fetal and Maternal Outcome, American Diabetes Association , Diabetes Care,2009.
42. Garner PR: Type 1 diabetes and pregnancy. Lancet 346:966, 1995
43. Yang J, Cummings EA, O'Connell C, et al: Fetal and neonatal outcomes of diabetic pregnancies. Obstet Gynecol 108:644, 2006
44. Hawthorne G: Maternal complications in diabetic pregnancy. Best Pract Res Clin Obstet Gynaecol 25(1):77, 2011
45. Gabbe SG, Graves CR: Management of diabetes mellitus complicating pregnancy. Obstet Gynecol 102:857, 2003
46. Gabbe S, Gregory R, Power M, et al: Management of diabetes mellitus by obstetricians-gynecologists. Obstet Gynecol 103:1229, 2004
47. Landon MB, Mele L, Spong CY, et al: The relationship between maternal glycemia and perinatal outcome. Obstet Gynecol 117(2):218,2011
48. Gabbe S, Gregory R, Power M, et al: Management of diabetes mellitus by obstetricians-gynecologists. Obstet Gynecol 103:1229, 2004

49. Kjos SL, Henry OA, Montoro M, et al: Insulin-requiring diabetes in pregnancy: a randomized trial of active induction of labor andexpectant management. Am J Obstet Gynecol 169:611, 1993
50. Narayan KM, Boyle JP, Thompson TJ, et al: Lifetime risk for diabetes mellitus in the United States. JAMA 290:1884, 2003 [PMID: 14532317

PROFORMA

Name : **Parity** :

Age : **IP No** :

Address : **D.O.A** :

History

- H/o. Polyuria
- H/o. Polydipsia
- H/o. excessive weight gain / weight loss
- H/o. Recurrent abortion
- H/o. Stillbirth
- H/o. Previous H/o. Glucose Intolerence
- H/o. Macrosomic Infant
- H/o.Congenital Anomelies in baby
- H/o. Type 2, Diabetes in 1st degree relatives – Father or Mother
- H/o. Auto immune disorders

H/o. Presenting illness

Menstrual History

Marital History

Past History

Family History

Personal History

GENERAL EXAMINATION

Ht BMI

Wt

Conscious

Afebrile

Anemia

Jaundice

Generalised lymphadenopathy

Clubbing

Acanthosis

Skin tags

Obstetric History

Previous Pregnancy : mode of delivery

H/o. Infertility

Conceived after Treatment

H/o. Abortion

H/o. PIH / Abruption

H/o. GDM

H/o. IUGR / LBW / PTL

Still birth

Baby weight admission in NICU

Present Pregnancy

Systemic Examination

Vital Signs

PR

BP

CVS

RS

Breast Thyroid

Spine Gait

Abdomen

P/A :

P/S :

P/V :

Urine : Albumin

Sugar

Deposit

Hemoglobin

GCT

GTT

HbA1C

RFT

Outcomes

Prematurity

IUD

Macrosomia

IUGR

Oligohydramnios

Shoulder dystocia

Fetus-hyper bilirubinemia

- Hypocalcemia
- Hypoglycemia
- Congenital anomalies

Mother

- PIH
- Antepartum Hemorrhage
- Oligohydramnios / polyhydramnios
- Preterm labour
- Caesarian deliveries
- Instrumental deliveries

MASTER CHART

S.NO	NAME	AGE	I D NO.	OBSTETRIC CODE	GESTATION AGE/WEEKS	HT	WT	BMI	GCT	GTT	H
1	JEYALAKSHMI	22	1077	G2P1L1	28 wk	150	49	19.6	68		
2	SHANTHIMARI	27	1079	G2P1L1	26 wk	146	50	23.5	64		
3	VINITHA	21	1084	G2P1L1	28wk	150	51	22.7	90		
4	PARAMESWARI	25	1086	G2P1L1	24wk	153	70	31.2	60		
5	**SRIDEVI**	**27**	1063	**G2P1L0**	24 wk	153	64	**26.9**	154	**106**	
6	ALAMMAL	30	1090	G2P1L1	26wk	148	52	23.7	90		
7	VIJI	29	1089	PRIMI	28wk	148	49	22.4	104		
8	PANDIAMMAL	22	1088	PRIMI	24wk	149	51	24.5	104		
9	CHITRADEVI	24	1085	G2P1L1	26WK	153	52	22.2	84		
10	MANJULA	24	1093	PRIMI	26wk	150	55	24.9	88		
11	BANUPRIYA	23	2797	G2P1L1	28wk	160	61	24.2	94		
12	REVATHI	28	1096	G4P3L1	26wk	160	61	30.4	102		
13	VIJAYALAKSHMI	28	1097	G2P1L1	24wk	150	51	22.7	92		
14	MEGALA	20	1394	PRIMI	24wk	153	70	30.1	68		
15	VASANTHI	32	12992	G3P2L2	26wk	160	72	28.12	83		
16	PANDEESWARI	23	1338	G2P1L1	26wk	151	52	23.4	82		
17	LAKSHMI	21	1337	PRIMI	28wk	147	53	24.5	104		
18	SHANTHI	34	1339	G3P1L1A1	26wk	149	41	18.5	106		
19	LAKSHMI	24	11364	G4P2L2A1	28wk	152	46	18.9	74		
20	JOTHI LAKSHMI	21	12587	PRIMI	26wk	146	48	22.5	82		
21	RANI	30	760	G2P1L1	28wk	156	61	23.2	86		
22	**Raman begam**	32	1054	**G3P2L2**	26 wk	152	66	**28.57**	148	**96**	
23	SELVI	33	13346	PRIMI	26wk	150	54	21.2	76		
24	INDIRA	30	1356	G2P1L1	28wk	158	60	22.1	68		
25	BACHIYALAKSHMI	22	1361	G2P1L1	26wk	160	54	19.1	135		
26	RANJITHA	20	1618	G2P1L1	28wk	146	50	18.7	75		
27	MUTHAMIL SELVI	20	1617	PRIMI	26wk	150	51	22.7	67		
28	THIRUPPATHI	24	1627	G2P1L1	28wk	152	49	21.2	77		
29	MUTHULAKSHMI	25	1621	PRIMI	28wk	152	46	19.9	96		
30	SAMSATH	26	1535	PRIMI	24wk	160	56	21.9	126		
31	SAKTHI	23	1624	PRIMI	26wk	155	53	22.1	78		
32	KALAIVANI	27	1626	PRIMI	25wk	150	51	22.7	76		
33	SUDHA	29	1002	G3P1L1A1	26wk	153	78	31.8	84		
34	CHINNAMANI	20	1344	PRIMI	20wk	163	51	19.2	107		
35	RAHHU	26	1008	G2P1L1	26wk	48	143	18.6	66		
36	SANTHI	34	1530	G3P2L2	28wk	158	49	19.6	108		
37	PRIYA	19	1939	PRIMI	28wk	151	53	23.2	69		
38	CHELLAMMAL	22	1938	G2P1L1	28wk	150	50	22.2	86		
39	RAJESWARI	21	1940	G2A1	26wk	155	59	24.6	92		
40	DHANALAKSHMI	38	1943	PRIMI	28wk	152	51	22.1	74		
41	MURUGESWARI	24	1948	G2P1L1	26wk	157	56	23.7	68		
42	SULTHANIA	24	2243	PRIMI	26wk	150	46	20.4	82		
43	**RAJESWARI**	24	1962	**Primi**	28wk	153	60	**25.64**	158	**102**	
44	JEYARANI	20	1524	PRIMI	26wk	148	54	24.7	65		
45	ANISHBEGAM	22	1266	G2P1L0	24wk	146	46	21.6	96		
46	SANTHANAMARI	21	2537	PRIMI	28wk	150	48	20.4	78		
47	**Shanthi**	31	2542	**G2P1L1**	26WK	160	74	**28.9**	162	**108**	
48	AMUTHA	32	2551	PRIMI	24wk	142	50	24.8	65		

49	NAGASUNDARI	19	2913	PRIMI	28wk	158	50	20.8	100		
50	**KALA**	**26**	2916	**G2A1**	26wk	152	61	**26.4**	154	**96**	
51	LAKSHMI	24	1391	G2P1L1	26wk	146	47	19.2	62		
52	MUNEESWARI	27	1392	G2P1L1	24wk	146	50	23.5	68		
53	CHINNAPONNU	26	1394	PRIMI	24wk	150	51	22.7	67		
54	MUTHULAKSHMI	27	1400	G2P1L1	26wk	149	49	21.2	94		
55	GEETHA	24	794	PRIMI	28wk	148	52	23.7	64		
56	KALAIYARASI	21	1650	PRIMI	25wk	160	56	24.5	104		
57	SANGEETHA	20	1652	PRIMI	26wk	151	52	18.7	85		
58	NITHYA	27	1653	G2P1L1	28wk	154	56	20.2	64		
59	PALANEESWARI	24	1654	G3P1L1A1	26wk	160	56	21.9	103		
60	VAISHNAVI	19	1656	PRIMI	28wk	150	52	22.7	94		
61	MUTHUMARI	18	1529	G2P1L1	25wk	147	39	18	62		
62	SRIDEVI	25	1663	G2P1L1	22wk	147	50	21.7	79		
63	AMALA	22	1664	PRIMI	26wk	152	58	22.6	79		
64	SRIDEVI	266	13366	G2P1L1	24wk	156	76	30.1	122		
65	SARASWATHI	22	824	PRIMI	26wk	146	56	24.1	91		
66	ALAGESWARI	25	1665	G2P1L1	28wk	162	59	22.1	68		
67	RADHA	25	1666	G2P1L1	26wk	153	54	20.1	82		
68	SANGEETHA	24	1667	PRIMI	24wk	149	56	21.4	123		
69	DHANAPANDIAMMAL	23	1659	PRIMI	26wk	151	49	20.2	118		
70	SARAL	21	1975	PRIMI	28wk	145	42	18.1	70		
71	**NANCY**	**26**	1662	**PRIMI**	26WK	156	58	**23.86**	154	**104**	
72	KURUVAMMAL	21	15819	G3P2L2	28wk	163	61	21.8	86		
73	PARKEES	24	1976	G3P2L2	26wk	166	67	20.6	74		
74	NILOFERNISHA	22	1974	PRIMI	28wk	156	58	20.8	65		
75	ALAGUMEENA	19	1913	PRIMI	28wk	160	64	24.1	68		
76	KRISHNAVENI	33	1633	G2P1L1	24wk	152	45	19.5	88		
77	SATHYA	26	1982	PRIMI	27wk	152	44	19	65		
78	PRIYA	25	1983	PRIMI	28wk	154	52	21.9	68		
79	KATHIRVELAMMAL	27	1644	G2P1L1	22wk	153	60	25.6	91		
80	MUTHU	24	1988	G2P1L1	26wk	156	57	53.4	97		
81	RAMZAN BEGAM	30	9537	G2P1L1	26wk	151	64	26.4	114		
82	PALMATHI	24	1443	G2P1L1	24wk	154	46	19.2	128		
83	RAJAPRIYA	22	1268	PRIMI	28wk	147	44	19.4	57		
84	MEENATCHI	26	1279	PRIMI	28wk	153	51	20.1	82		
85	ALAGAMMAL	36	1126	G2P2L2	26wk	141	58	29.2	49		
86	SELVARANI	21	2280	PRIMI	25wk	152	48	18.7	81		
87	**ASTALAKSHMI**	**27**	15814	**G2P1L1**	24wk	143	57	24.21	**146**	**98**	
88	MUMITHA BEGAM	28	2281	G2P1L1	28WK	160	62	**22.6**	**60**		
89	THEERTHAM	24	2288	G2P1L1	26wk	142	46	22.8	58		
90	DHANALAKSHMI	20	850	PRIMI	28wk	150	50	22.2	75		
91	VALLI	22	2605	G2P1L1	28wk	155	59	24.6	87		
92	SABIRA	23	2604	PRIMI	28wk	152	51	22.1	95		
93	MANIMEGALAI	20	2603	G2P1L1	26wk	148	62	28.3	72		
94	PONNATHAL	23	2607	G2P1L1	24wk	157	56	22.7	66		
95	IRULAYEE	34	2606	G2P1L1	25wk	148	47	21.5	89		
96	MAHESWARI	21	1486	G4P3L2	27wk	150	46	20.4	80		
97	NOORIBEGAM	29	8943	G2P1L1	29wk	156	66	28.2	102		
98	ARTHI	20	2616	G4P3L3	28wk	149	52	23.4	107		
99	**MUNEESWARI**	**28**	1267	**G2P1L1**	26WK	149	51	**30.4**	**170**	**109**	
100	MURUGESWARI	25	2042	G2P1L1	28wk	156	52	24.2	124		
101	PRABA	20	1274	G2P1L1	24wk	148	48	21.9	135		
102	SOUNDARYA	19	1276	PRIMI	28wk	158	52	20.8	131		

103	SARASWATHI	23	1546	G3P2L2	26wk	156	62	25.5	70		
104	VENGAYEE	21	1389	G2P1L1	27wk	152	56	24.2	64		
105	ESWARI	29	1279	G3P1L1A1	25wk	150	48	21.3	72		
106	ALAGUSUNDARI	32	1296	PRIMI	26wk	156	60	24.7	84		
107	MEENATCHI	28	1456	PRIMI	28wk	148	48	21.9	92		
108	BANUPRIYA	23	1761	G2P1L1	26wk	158	52	20.8	86		
109	KALAISELVI	27	1762	G3P2L2	25wk	146	46	21.6	117		
110	SUGANYA	23	1534	PRIMI	22wk	156	62	25.5	121		
111	SHANTHI	24	9532	G2P1L1	24wk	156	57	24.9	122		
112	MUTHUPANDI	20	1584	G3A2	24wk	148	46	20.1	90		
113	SEVATHAMAL	21	1507	G2P1L1	26wk	148	54	24.7	100		
114	***SELVI***	26	1564	PRIMI	28WK	153	54	21.7	**140**	**86**	
115	RAMALAKSHMI	21	1571	G2P1L1	28wk	159	56	22.4	108		
116	GNANAVALLI	24	1507	PRIMI	24wk	164	63	23.4	108		
117	MEENA	35	1587	G4P3L3	26wk	146	48	21	92		
118	KALA	28	8630	G2P1L1	28wk	152	54	24.8	95		
119	SUMATHI	28	245	G2P1L1	28wk	152	50	21.2	84		
120	ALAGUSUNDARI	24	1528	PRIMI	27wk	159	56	22.1	90		
121	ABINAYA	20	1639	PRIMI	25wk	160	61	26.4	122		
122	SATHYA	19	1327	G3P2L2	28wk	161	77	29.7	91		
123	RAJESWARI	21	1515	PRIMI	28wk	142	49	20.4	121		
124	VAIDEHI	20	1844	PRIMI	26wk	161	50	19.3	91		
125	SARATHI	23	1846	PRIMI	25wk	155	50	20.8	121		
126	SUMATHI	23	1843	G2P1L1	24wk	152	50	21.6	91		
127	PANDIMEENA	27	1847	PRIMI	24wk	140	45	19.3	51		
128	DEEPIKA	19	1543	G2P1L1	25wk	157	60	23.4	87		
129	KRISHNAVENI	27	1849	G3P1L1A1	22wk	152	50	21.6	95		
130	SANKARESWARI	22	1852	G2P1L1	28wk	161	50	19.3	60		
131	PAVITHA	26	1858	G2P1L1	24wk	142	45	22.3	95		
132	POOMARI	20	1860	PRIMI	28wk	150	56	24.4	68		
133	MALAR	24	1863	G2P1L1	26WK	162	66	24.3	**142**	84	
134	REVATHI	23	1871	G2P1L1	24wk	150	63	28	75		
135	SANGEETHA	22	1880	PRIMI	28wk	150	53	23.6	115		
136	SELVALAKSHMI	25	1851	PRIMI	26wk	143	53	22.5	110		
137	SATHYAPRIYA	21	1566	PRIMI	24wk	148	46	19.4	110		
138	ATHELAHSMI	29	1872	G3P2L1	28wk	156	62	25.5	60		
139	SASIKALA	29	1873	G3P2L2	26wk	152	62	26.8	77		
140	NOORSA	24	2181	G2P1L1	22wk	150	55	24.1	84		
141	MUTHUMARI	28	2193	PRIMI	28wk	152	55	23.8	77		
142	MANIKAPRIYA	20	2194	G3P2L2	26wk	158	73	29.2	77		
143	GAYATHRI	23	2195	G2P1L1	25wk	151	44	19.9	117		
144	**JEYA**	22	1913	PRIMI	26WK	148	51	20.64	**148**	**90**	
145	SANGEETHA	20	1914	PRIMI	28wk	156	55	22.6	110		
146	BHUVANESWARI	25	2181	PRIMI	24wk	141	42	20.3	60		
147	VASANTHI	26	2184	PRIMI	26wk	160	67	26.2	82		
148	MEENATCHI	30	2193	G4P3L2	28wk	159	50	19.8	77		
149	PANDEESWARI	26	2195	G2P1L1	22wk	161	50	19.3	84		
150	MANIMEGALAI	24	1914	PRIMI	26wk	142	50	24.8	77		
151	KALEESWARI	24	2180	PRIMI	28wk	155	50	20.8	77		
152	VEERALAKSHMI	20	2985	PRIMI	25wk	152	50	21.6	117		
153	RADHA	30	2453	G3P1L1A1	27wk	148	46	21	101		
154	SYETHALIFATHIMA	27	2451	G2P1L1	22wk	144	40	19.3	115		
155	ALAGUPANDIAMMAL	22	2450	G2P1L1	25wk	157	65	26.4	96		
156	CHANDRA	25	2455	PRIMI	26wk	159	68	26.1	89		

157	JEYAKALA	26	2454	PRIMI	28wk	150	52	21.6	91		
158	MAHALAKSHMI	28	2458	G2P1L1	25wk	148	46	21	88		
159	MANIMEGALAI	25	2460	G2P1L1	23wk	150	48	17.6	102		
160	HEMALATHA	23	2471	G2A1	26wk	152	50	19.6	114		
161	KULANTHAIAMMAL	26	2459	PRIMI	26wk	156	50	23.3	84		
162	**RASATHI**	**29**	2461	G2A1	28wk	156	57	24.6	**140**	**82**	
163	NALENA	23	2471	PRIMI	24wk	147	50	24.4	82		
164	CHINNATHAI	20	2852	PRIMI	26wk	161	56	21.6	68		
165	MUTHU	26	2651	PRIMI	28WK	154	62	27.2	106		
166	VIJAYA	28	2662	PRIMI	26wk	160	62	22.4	80		
167	KARTHEESWARI	24	1289	G2P1L1	22wk	142	45	19.7	80		
168	VISNUDEVI	21	1671	PRIMI	25wk	155	45	18.7	76		
169	KANNIKADEVI	20	2854	PRIMI	28wk	155	51	24.1	89		
170	KATHAMMAL	23	2857	G2P1L1	28wk	148	48	26.5	84		
171	SANKARESWARI	22	2881	G2P1L1	26wk	151	55	24.1	70		
172	CHELLAMMAL	20	2887	G2P1L1	24wk	161	52	20.4	66		
173	MURUGESWARI	22	1426	G2P1L1	28wk	155	53	20.4	119		
174	CHITTU	32	1274	G2P1L0	26WK	148	59	24.9	128		
175	PONMANI	22	1519	G2P1L1	24wk	149	51	20.1	81		
176	MUTHU	27	3151	G2A1	26wk	162	64	22.2	73		
177	ALAGU	24	3157	G2P1L1	26WK	152	56	23.42	82		
178	KARTHIGA	30	3152	G2P1L1	24wk	149	46	19.1	65		
179	ANANTHI	24	3153	G2P1L1	24wk	151	54	20.1	68		
180	RAJESWARI	20	3162	G2P1L1	24wk	160	67	25.4	66		
181	JEYARANI	32	3161	G2P1L1	26wk	150	51	21.9	119		
182	PANVATHI	29	3170	G2P1L1	24wk	155	48	20	81		
183	VASANTHI	32	12992	G2P1L1	24wk	153	63	26.9	74		
184	KARTHIGA	19	2216	G2P1L1	28wk	150	48	19.1	61		
185	KARTHIGAIPRIYA	23	3165	G2P1L1	22wk	152	56	24.2	73		
186	MEENA	21	3168	G2P1L1	24wk	161	77	30.4	80		
187	MURUGESWARI	23	3173	G2P1L1	26wk	160	60	23.4	84		
188	PARKATHNISHA	23	2875	G2P1L1	28wk	142	59	29.3	64		
189	KALEESWARI	21	2846	PRIMI	26wk	155	51	21.2	72		
190	MAHALAKSHMI	24	1876	PRIMI	28wk	148	40	21	139		
191	VIJI	25	3476	PRIMI	28wk	161	50	19.3	99		
192	KALEESWARI	28	4321	G2P1L1	26wk	155	50	20.8	98		
193	IDAYAKANI	20	4310	G2P1L1	22wk	145	45	21.4	98		
194	SIVANESWARI	24	2497	G2P1L1	24wk	144	40	19.3	88		
195	BANUMATHI	20	3492	G2P1L1	22wk	155	49	20	92		
196	ANGALAESWARI	21	2498	G2P1L1	28wk	161	51	20.1	114		
197	BHARATHI	23	2510	G2P1L1	24wk	157	65	30.6	98		
198	MUTHUSELVI	20	3495	G2P1L1	26wk	149	58	26.1	116		
199	MANIMEGALAI	29	2512	G3P2L1	24wk	157	61	25.8	66		
200	TAMILARASI	23	2497	G2P1L1	26wk	142	59	29.3	80		

RISK FACTORS IN PAST PREGNANCY	RISK FACTORS IN PRESENT PREGNANCY	MODE OF DELIVERY	C
		LABOUR NATURAL	
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		LABOURNATURAL WITH EPISIOTOMY	
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		LABOURNATURALWITHEPISIOTOMY	
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		ELECTIVELSCS	
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SPONTANEOUS ABORTION		LABOURNATURALWITHEPISIOTOMY	
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preeclampsia		**LABOUR NATURAL**	
	PRE ECLAMPSIA	LABOUR NATURAL	
		OUTLETFORCEPS	
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		ELECTIVELSCS	
SPONTANEOUS ABORTION		LABOURNATURALWITHEPISIOTOMY	
Spontaneous abortion		**EMERGENCY LSCS**	HYP

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	PREECLAMPSIA	LABOURNATURAL	
	PRE ECLAMPSIA	OUTLETFORCEPS	
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	OBESITY,PREECLAMPSIA	**LABOUR NATURAL**	
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SPONTANEOUS ABORTION	PRE ECLAMPSIA	ELECTIVELSCS	
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SPONTANEOUS ABORTION		EMERGENCYLSCS	
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Ref. No1864/E4/2/2014,

Govt. Rajaji Hospital,
Madurai.20. Dated: 29.03.2014

Institutional Review Board / Independent Ethics Committee.
Capt. Dr.B. Santhakumar, M.D., (F.M.,) deanmdu@gmail.com
Dean, Madurai Medical College &
Govt Rajaji Hospital, Madurai 625020. **Convenor**

Sub: Establishment-Govt. Rajaji Hospital, Madurai-20-
Ethics committee-Meeting Minutes- for March 2014
Approved list - Regarding.

The Ethics Committee meeting of the Govt. Rajaji Hospital, Madurai was held on 05.03.2014, Wednesday at 10.00 am to 12.00.noon at the Auditorium, Govt. Rajaji Hospital, Madurai. The following members of the committee have attended the meeting.

1.Dr.V. Nagarajan, M.D., D.M (Neuro) Ph: 0452-2629629 Cell.No 9843052029 ndg9999@gmail.com	Professor of Neurology (Retired) D.No.72, Vakkil New Street, Simmakkal, Madurai -1	Chairman
2. Dr.Mohan Prasad , M.S M.Ch Cell.No.9843050822 (Oncology) drbkcmp@gmail.com	Professor & H.O.D of Surgical Oncology(Retired) D.No.32, West Avani Moola Street, Madurai -1	Member Secretary
3. Dr. Parameswari M.D (Pharmacology) Cell.No.9994026056 drparameswari@yahoo.com	Director of Pharmacology Madurai Medical College	Member
4. Dr.S. Vadivel Murugan, MD., (Gen.Medicine) Cell.No 9566543048 svadivelmurugan_2007@rediffmail.com	Professor& H.O.D of Medicine Madurai Medical College	Member
5. Dr.S. Meenakshi Sundaram, MS (Gen.Surgery) Cell.No 9842138031 drsundarms@gmail.com	Professor & H.O.D of Surgery Madurai Medical College	Member
6. Mrs. Mercy Immaculate Rubalatha, M.A., Med., Cell. No. 9367792650 lathadevadoss86@gmail.com	50/5, Corporation Officer's quarters, Gandhi Museum Road, Thamukam, Madurai-20	Member
7. Thiru..Pala. .Ramasamy , BA.,B.L., Cell.No 9842165127 palaramasamy2011@gmail.com	Advocate, D.No.72.Palam Station Road, Sellur, Madurai -2	Member
8. Thiru. P.K.M. Chelliah ,B.A Cell.No 9894349599 pkmandco@gmail.com	Businessman, 21 Jawahar Street, Gandhi Nagar, Madurai-20	Member

The following Projects was approved by the committee.

Name of P.G.	Course	Name of the Project	Remarks
Dr. V. Poomatha, vpoomatha@gmail.com	PG in M.S., (O&G) Madurai Medical College and Government Rajaji Hospital, Madurai.	Case Control Study on Prevalence of Gestational Diabetes Mellitus during First and Second Trimester of Pregnancy and their obstetric outcomes.	Approved

Please note that the investigator should adhere the following: She/He should get a detailed informed consent from the patients/participants and maintain it Confidentially.

1. She/He should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution or to Government.
2. She/He should inform the institution Ethical Committee, in case of any change of study procedure, site and investigation or guide.
3. She/He should not deviate the area of the work for which applied for Ethical clearance. She/He should inform the IEC immediately, in case of any adverse events or Serious adverse reactions.
4. She/He should abide to the rules and regulations of the institution.
5. She/He should complete the work within the specific period and if any Extension of time is required He/She should apply for permission again and do the work.
6. She/He should submit the summary of the work to the Ethical Committee on Completion of the work.
7. She/He should not claim any funds from the institution while doing the work or on completion.
8. She/He should understand that the members of IEC have the right to monitor the work with prior intimation.

Member Secretary **Chairman**
Ethical Committee

29.3.14
DEAN/Convenor
Govt. Rajaji Hospital,
Madurai- 20.

19/3/14

To
The above Applicant
-thro. Head of the Department concerned

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CASE CONTROL STUDY ON PREVALENCE OF
GESTATIONAL DIABETES MELLITUS IN FIRST AND
SECOND TRIMESTER OF PREGNANCY AND
ITS OBSTETRIC OUTCOMES

A dissertation submitted to the

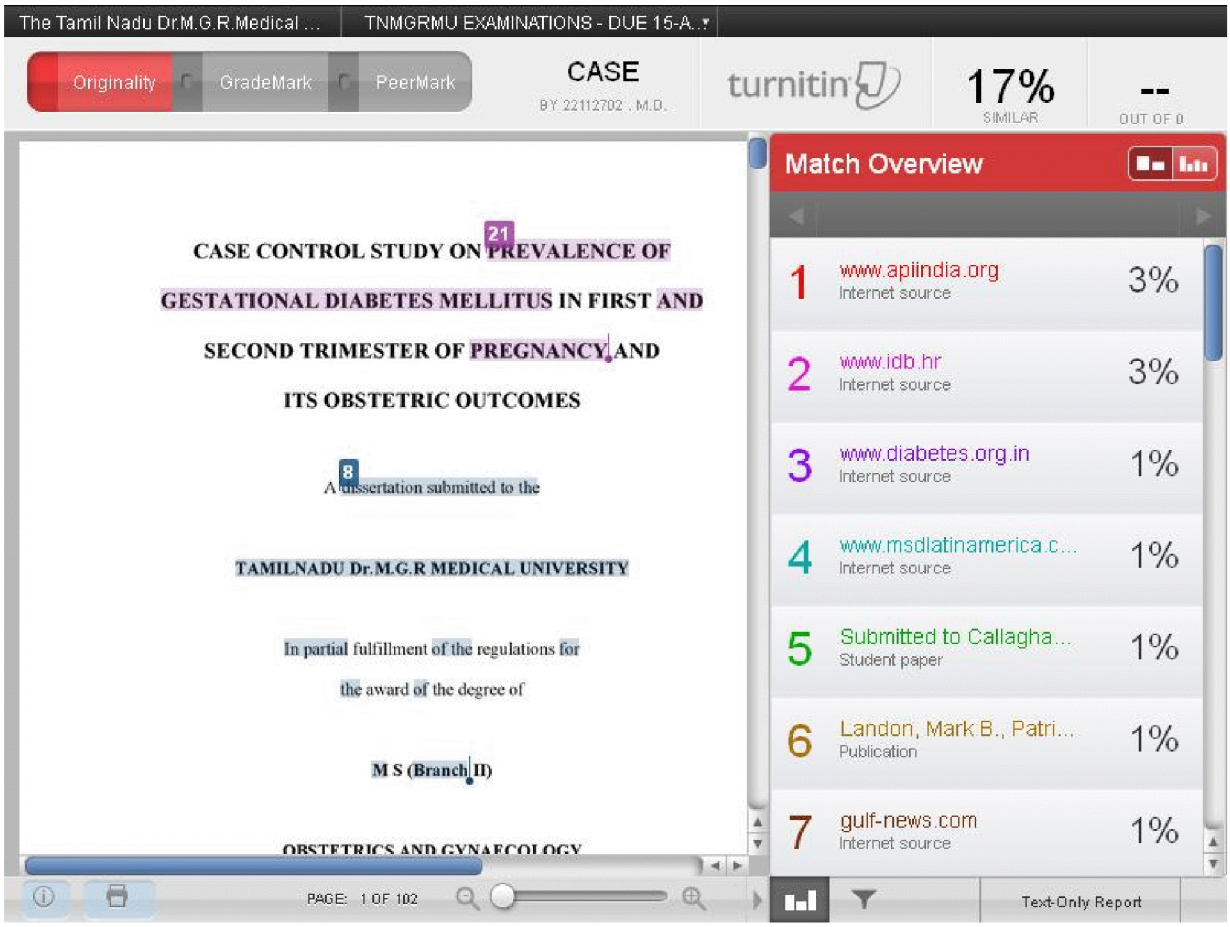
TAMILNADU Dr.M.G.R MEDICAL UNIVERSITY

In partial fulfillment of the regulations for
the award of the degree of

M S (Branch II)

OBSTETRICS AND GYNAECOLOGY

MADURAI MEDICAL COLLEGE
MADURAI – 625014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ITS OBSTETRIC OUTCOMES
A dissertation submitted to the
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